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THE ETIOLOGY OF PNEUMONIA IN THE PIG.

By J. M'FADYEAN, Royal Veterinary College, London.

IN an article which appeared in this Journal for September last, reference was made to the discrepant views held by different veterinary surgeons regarding the etiology and diagnostic value of pneumonic lesions encountered in pigs suspected of swine-fever, and readers of the Journal were requested to forward for examination pig's lungs showing the lesions of pulmonary inflammation. During the last three months of 1896 less than a dozen sets of diseased lungs were received at the Research Laboratory, and the fact may be adduced as evidence of the comparative rarity of pneumonia in pigs affected with swine-fever. Although the number of specimens received was thus small, it was not found possible to carry out the projected experiments and observations with each of them, for, as the following account will show, the investigation of even one such case involves the expenditure of a good deal of time and trouble. A few of the cases were selected for examination, and the results will be described in the following pages.[1]

CASE NO. I.

The parts received for examination in this case were a piece of large intestine showing the characteristic swine-fever ulceration, and a piece of lung with an area of firm hepatisation the size of a hen's egg. The pig had been killed on the morning of the day on which the parts were received at the laboratory. A cover-glass preparation made from the hepatised area, and stained with methyl blue, showed fairly numerous short bacteria, and slanting agar tubes inoculated from the lesion yielded, after twenty-four hours' incubation at 37° C., numerous circular, flat, transparent colonies, which on microscopic examination were found to be composed of short ovoid bacteria resembling those of fowl cholera. When re-inoculated into bouillon the organism grew in that medium as a fluffy growth at the bottom of the tube, the liquid remaining clear. In hanging-drop preparations the organism was found to be non-motile, and in gelatine-agar medium it formed spherical colonies, easily distinguished from those of the swine-fever bacillus. No growth took place on potato.

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[1] This opportunity is taken to return cordial thanks to the following gentlemen for their kindness in forwarding specimens of the nature alluded to:—Messrs J. D. Armfield, Oldham; G. E. King, Abingdon; G. W. M. Haydon, Midsomer Norton; T. B. Bindloss, Long Sutton; T. M. M'Connell, Wigtown; H. Kidd, Melton Mowbray; W. Anderson, Maybole; H. Edgar, Wanstead; J. W. Edwards, Kingston-on-Thames; J. F. Simpson, Maidenhead.

Two rabbits (A and B) were subcutaneously inoculated on the abdomen with a scraping taken from the centre of the hepatised area in the pig's lung. The scraping was suspended in bouillon, and the experiment was made with the customary precautions.

One of the rabbits (A) was found dead forty-eight hours after inoculation. The *post-mortem* examination showed a very slight inflammatory reaction at the seat of inoculation. A cover-glass preparation made from the blood of the ear showed fairly numerous short bacteria. Slanting agar tubes inoculated from the heart yielded an apparently pure culture of short bacteria with the characters above described.

The second rabbit (B) was found dead on the fourth day after inoculation. The subcutaneous tissue over almost the entire abdominal wall was the seat of inflammatory congestion and œdema, with some blanching of the tissues from incipient necrosis. The peritoneum was intensely inflamed, and covered with flakes of fibrinous lymph. A preparation from the ear blood showed a few short or ovoid bacteria, and slanting agar tubes inoculated from the heart yielded these in apparently pure culture.

Inoculated a rabbit (C) subcutaneously on the abdomen with 1 cc. bouillon culture, being the first generation from the above rabbit B. The culture had been incubated for forty-eight hours at 37°, after which it had remained for five days at the room temperature. The bouillon was very slightly turbid, and a fluffy growth was present at the bottom of the tube.

The rabbit was found dead on the fourth day after inoculation. At the seat of inoculation, and spreading over the greater part of the abdominal wall, there was an inflammatory lesion. The peritoneum was the seat of dry inflammation, and the intestinal loops in contact with the abdominal wall were matted together with fibrinous lymph. A cover-glass preparation made from the ear blood showed only a small number of short ovoid bacteria, and from the heart these were recovered in apparently pure culture on slanting agar.

A rabbit (D) was inoculated subcutaneously on the abdomen with a culture which was the second generation from the last-named rabbit (C), and at the same time a pigeon was inoculated into the pectoral muscles.

The rabbit was found dead on the third day after inoculation. A cover-glass preparation from the blood of the ear showed great numbers of short bacteria. At the seat of injection on the abdominal wall there was a lesion about 2½ inches in diameter; the tissues here were swollen and œdematous. The peritoneum was inflamed, and the intestines were matted together with fibrinous lymph. The pleura was also inflamed on both sides, and entirely covered with a thin greyish layer of lymph. The pericardium was in a similar condition. Cover-glass preparations made from the fibrinous lymph showed immense numbers of short bacteria. The organism was recovered in apparently pure culture on slanting agar and boullion tubes inoculated from the pleural lymph, and from the heart blood.

The pigeon was found dead on the fifth day after inoculation. At the seat of inoculation in the right pectoral muscle there was a blanched area of necrosis larger than a hazel nut. Cover-glass preparations made from the blood showed only a small number of ovoid bacteria, free and included in leucocytes. The organism was recovered in apparently pure culture on slanting agar and in bouillon inoculated from the heart, and from the local lesion in the pectoral muscle.

Inoculated a rabbit (E) subcutaneously on the abdomen with a culture which was the first generation from the pectoral muscle of the above-mentioned pigeon.

The rabbit was found dead on the fourth day after inoculation, with an extensive lesion at the seat of inoculation and peritonitis with a dry fibrinous exudate. Slanting agar and bouillon tubes inoculated from the heart blood and from the local lesion yielded abundant apparently pure growth.

The carcase of the rabbit (E) of the last experiment was skinned and decapitated; it was then cut into pieces and given along with the viscera to a pig (A) about six months old. The pig had been starved for some time previously, and the whole of the flesh and the greater part of the viscera were consumed.

This pig was killed on the seventh day of the experiment. During the interval it had not shown any symptom of ill-health. The only abnormalities discovered at the *post-mortem* examination were the presence of some spots of congestion on the mucous membrane of the colon, and intense general congestion of the mucous membrane of the ileum. In neither large nor small intestine was there any trace of ulceration or diphtheritic deposit. Slanting agar tubes inoculated from the mesenteric glands remained sterile.

Inoculated a pig (B) subcutaneously in the right thigh with 1½ cc. of sterile water holding in suspension part of a slanting agar culture, which was the second generation from the last-mentioned rabbit (E). At the same time 4 cc. of the same liquid were injected through the left chest wall of another pig (C) of the same lot (about six months old).

The pig inoculated subcutaneously (B) was killed on the tenth day of the experiment. At the seat of inoculation in the right thigh there was a subcutaneous nodule as large as a horse bean. Several of the mesenteric glands were enlarged, and on section one of them was found to be caseating.[1] No other lesions were discovered. Two slanting agar tubes from the right thigh yielded an abundant apparently pure growth of the organism used for inoculation.

The pig (C) inoculated through the chest wall was also killed on the tenth day of the experiment. The seat of inoculation was not discoverable either in the textures of the thoracic wall, in the pleura, or in the lung. Several of the mesenteric glands were found to be indurated and caseating. No other lesion was discovered.

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[1] This and the other pigs in which tuberculous lesions were found in the mesenteric glands belonged to the same litter. They had apparently been infected with tuberculosis before they were purchased for experiment.

CASE No. II.

The parts received for examination in this instance were a piece of large intestine with typical swine-fever ulcers, and the left lung of the same pig. The apex of the lung and part of the lower edge of its main lobe behind the heart were hepatised. The hepatised part had a mottled surface—greyish pink with streaks of opaque yellowish-white suggestive of necrosis. The pig had died on the morning of the day previous to that on which the parts were received at the laboratory. A cover-glass preparation made from the hepatised part showed only a sparing number of short or ovoid bacteria.

Two rabbits (A and B) were inoculated subcutaneously on the abdomen with a scraping from the hepatised part suspended in bouillon.

One rabbit (A) was found dead on the seventh day after inoculation. Spreading from the seat of inoculation over almost the entire abdominal wall there was an inflammatory lesion associated with a good deal of necrosis.

At its maximum the abdominal wall was four or five times its normal thickness, and on its peritoneal side it was covered with yellow fibrinous lymph. A cover-glass preparation made from the blood of the ear showed no free bacteria, but very numerous purple granules in the leucocytes (methyl-blue staining). Two slanting agar tubes inoculated from the heart blood yielded each about twenty discrete colonies, and a similar but more abundant growth was obtained on two slanting agar tubes inoculated from the lesion of the abdominal wall.

The second rabbit (B) was found dead on the eleventh day after inoculation. As in the previous case, there was a very extensive lesion of the abdominal wall associated with some necrosis of the tissues. No peritonitis. No free bacteria in the blood of the ear, but numerous violet granules in the leucocytes (methyl-blue staining). A slanting agar tube inoculated from the abdominal wall yielded an almost continuous transparent growth, and two slanting agar tubes inoculated from the heart blood yielded each about a dozen colonies. These colonies were flat, circular, and translucent, and on examination they were found to be composed of bacteria of the fowl cholera type. The organism was non-motile, and not distinguishable, morphologically, or by its mode of growth, from the bacterium isolated from Case I.

A rabbit (C) was inoculated subcutaneously on the abdomen with a culture which was the second generation from the above-mentioned rabbit (A), and with the same culture a pigeon was inoculated at the same time into the pectoral muscle.

The rabbit was found dead on the eleventh day after inoculation. A cover-glass preparation made from the blood of the ear showed no bacteria. At the seat of inoculation and extending over nearly the whole of the right half of the abdominal wall there was an inflammatory lesion associated with much thickening of the textures of the abdominal wall, and a good deal of necrosis. At some places the textures of the abdominal wall had been broken down so as to resemble thick tenacious pus. A slanting agar tube inoculated with a loopful of blood from the heart remained sterile, but two slanting agar tubes inoculated from the local lesion yielded each an abundant growth in the form of flat, circular, transparent colonies.

The above pigeon was alive and apparently healthy two months after inoculation.

Inoculated a rabbit (D) subcutaneously in the groin with 5 cc.[1] mixed bouillon and slanting agar cultures, each being the first generation from the last-mentioned rabbit.

The rabbit was found dead on the third day after inoculation. The *post-mortem* examination showed an extensive inflammatory lesion starting from the seat of inoculation, as well as pleurisy and pericarditis with flakes of fibrinous lymph, but no liquid exudate. The organism was recovered in apparently pure culture on slanting agar tubes inoculated from the blood, inoculation lesion, and pleural lymph.

The rabbit used in the last experiment (D), after skinning and decapitation, was cut into pieces and given together with the viscera to a pig (A) about six months old. The pig had been starved for some time previously, and the whole was greedily consumed by it.

The pig was killed on the seventh day after the beginning of the experiment. The mucous membrane of the ileum showed intense general congestion, and some spots of congestion were also present in the large intestine. No trace of ulceration or diphtheritic deposit was present in any part of the alimentary canal, and no lesion except the above-mentioned congestion was found in any of the organs. Slanting agar tubes inoculated from the mesenteric glands remained sterile.

By the aid of a platinum needle a culture on slanting agar (second generation from the blood of rabbit D) was detached into 10 cc. of sterile bouillon, and 1½ cc. of this liquid was used to inoculate a pig (B) subcutaneously in the right thigh, while about 4 cc. of the same liquid were injected into another pig (C) through the left chest wall. Both pigs were about six months old.

On the morning of the day after inoculation, the pig inoculated in the thigh (B) was found to be very ill, and it died twenty-two hours after inoculation. The *post-mortem* examination revealed the following lesions:—

At the seat of inoculation there was a lesion about the size of a hazel nut; this was firm and on section rather dry, and reddish-yellow in colour. There was general peritonitis with abundant fibrinous lymph matting the intestines and causing easily separated adhesions between the organs and the parietal peritoneum. No appreciable liquid in the peritoneum. Pleurisy and pericarditis without liquid, but with the same fibrinous lymph. Throughout both large and small intestines there were irregular areas of congested mucous membrane, and the large Peyer's patches below the ileo cæcal valve had a livid colour. The lungs, stomach, spleen, liver, kidneys, tongue, and throat were normal.

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[1] A large dose was here intentionally used with the object of killing the rabbit with the septicæmic type of the disease.

No organisms were discoverable in cover-glass preparations made from the inflammatory exudate. Slanting agar tubes inoculated from pleural lymph, peritoneal lymph, pericardial lymph, and spleen pulp all remained sterile. A bouillon tube inoculated with a loopful of blood from the heart also remained sterile. Slanting agar tubes inoculated from the lesion in the right thigh yielded an abundant apparently pure growth of the organism used for inoculation.

The cause of this pig's illness, and of the peritonitis, pleurisy, and pericarditis of which it was the subject, remains obscure. It was at first thought that these lesions were ascribable to the organism used for inoculation, but that conclusion does not seem admissible in face of the failure to find these organisms on microscopic examination of the inflammatory lymph, or to demonstrate their presence by culture. The conclusion is also negatived by the result of the *post-mortem* examination of the other pig.

The pig inoculated through the chest wall (C) was killed on the ninth day of the experiment. In the interval it had not shown any symptom of ill health. *Post-mortem* examination showed along the line of the ninth rib on the left side a firm adhesion between the lung and the parietal pleura, but there was no consolidation of the lung at this point or elsewhere. Other organs normal.

CASE NO. III.

The parts received for examination in this case were both lungs and the cæcum. The pig was said to have been the subject of swine fever, but no distinct lesions were present in the piece of bowel sent. In the right lung there was a large area of hepatisation opposite the heart; on section the solid part showed a pale pink ground surface beset with yellowish-white tubercle-like points, varying in size from a pin's head to a barley grain. The bronchi in this part contained a thin muco-purulent liquid. The bronchial and tracheal glands were much enlarged, but on section showed no caseation. Tubercle bacilli could not be found either in scrapings from the lung lesion or from the glands. Nearly the whole of the left lung, with the exception of the large lobe, was hepatised, and on section the solid part showed the appearances above described.

Two rabbits were inoculated, one (A) subcutaneously and the other (B) intraperitoneally, with bouillon holding in suspension a scraping from the lung lesion of the above pig. Both rabbits were found dead the following day, less than twenty-four hours after inoculation.

In the rabbit (A) there was an inflammatory swelling 2 inches in diameter at the seat of inoculation in the abdominal wall. All the internal organs appeared normal, with the exception of the liver, which contained some spots of psorospermosis. Cover-glass preparations from the blood of the ear showed great numbers of short or ovoid bacteria. Two slanting agar tubes inoculated from the heart yielded, after twenty-four hours' incubation at 37° C., an abundant crop of flat, transparent, circular colonies composed of bacteria of the fowl cholera type. Two bouillon tubes inoculated each with a loopful of blood were slightly turbid after twenty-four hours with numerous coarse flocculi suspended in the liquid; with continued incubation the bouillon soon became clear, and deposited a coarse, flocculent precipitate.

In rabbit (B) the peritoneum was not inflamed, but at the point where the needle had been inserted there was a small cyst-like structure under the peritoneum, as if the fluid had been injected there and not into the peritoneal cavity. All the organs appeared to be normal, with the exception of the liver, which contained some spots of psorospermosis.

An organism apparently identical with that above described was obtained on slanting agar and in bouillon tubes from the heart.

The organism cultivated from these two rabbits was found to have characters similar to the bacteria isolated from Cases I. and II. It was non-motile and did not grow on potato. On slanting agar it grew in the form of circular, flat, transparent colonies. In gelatine-agar the discrete colonies were spherical or oval, with a well-defined periphery. Bouillon cultures remained almost clear, with coarse suspended particles, which subsequently settled as a bottom growth.

Inoculated a rabbit (C) subcutaneously on the abdomen with ½ cc. of mixed bouillon and slanting agar cultures, each being the second generation from one of the above-mentioned rabbits. At the same time a pigeon was inoculated into the pectoral muscle with ½ cc. of the same liquid.

Both the rabbit and the pigeon were found dead on the following day, less than twenty-four hours after inoculation. At the seat of inoculation in the rabbit there was an area of inflammation, with injection of the vessels and œdema of the tissues. Cover-glass preparations from the blood of the ear showed immense numbers of short and ovoid bacteria. No visceral lesions.

Around the seat of inoculation in the pectoral muscle of the pigeon there was considerable blood extravasation. No visceral lesions. Bacteria numerous in the blood.

From heart blood of both pigeon and rabbit the organism was recovered in apparently pure culture on slanting agar and in bouillon.

Inoculated a rabbit (D) subcutaneously on the abdomen with 1 cc. of bouillon culture, which was the fourth generation from the heart of rabbit A.

The rabbit was found dead on the following day, eighteen hours after inoculation. A cover-glass preparation from the blood of the ear showed enormous numbers of short bacteria.

After having been skinned and decapitated the carcase of the above rabbit was cut up into pieces, and given together with the viscera to a pig (A) about six months old. Over the pieces of carcase and viscera 48 cc. of a bouillon culture, which had been incubated for forty hours at 37° C., were sprinkled. This bouillon culture was the fourth generation from the heart of rabbit (A). The pig had been starved for some time previously, and the whole was greedily consumed by it.

On the second day of the experiment this pig had profuse diarrhœa; on the third day the fæces were normal; on the fourth day there was again some diarrhœa. Throughout the experiment the pig fed well and appeared quite lively. It was killed on the seventh day of the experiment. The mucous membrane of the colon was at some places diffusely congested, and its surface appeared slightly eroded, but there was no trace of diphtheritic deposit. The cæcum and the rest of the alimentary canal were normal, as were also all the other organs of the abdomen and thorax. Two slanting agar tubes inoculated from the mesenteric glands remained sterile.

Took a slanting agar culture, which was the third generation from the heart blood of the before-mentioned pigeon, and had been incubated for five days at 38° C.; poured into this about 8 cc. of sterile water and detached the surface growth by means of a platinum needle. Inoculated a pig (B) subcutaneously in the right thigh with 1½ cc. of this liquid, and at the same time injected 4 cc. of the liquid through the left chest wall of another pig (C). Both pigs were about six months old.

Pig (B) was killed on the ninth day of the experiment. In the interval it had not shown any symptom of ill health. At the seat of inoculation in the right thigh there was a swelling about the size of a pigeon's egg; centrally this was found to contain thick pus and broken down tissue. Some of the mesenteric lymphatic glands were enlarged, indurated, and commencing to caseate (tuberculous). No other lesions. Two slanting agar tubes inoculated from the lesion in the thigh yielded each an abundant apparently pure growth.

Pig (C) (inoculated through the chest wall) was also killed on the ninth day of the experiment; it had appeared to be quite well during the interval. The *post-mortem* examination showed that the left lung was partially adherent to the chest wall, and where the lung was free the left pleural sac contained a quantity of reddish turbid liquid. The free surface of the pleura was covered with a thick layer of yellow fibrinous lymph. There was also an adhesion between the lower edge of the right lung and the chest wall. The left lung was partly collapsed from pressure, but there was no pneumonic lesion in either lung. A small crater-like (tuberculous) ulcer was present in the small intestine, and two of the mesenteric lymphatic glands were enlarged and caseating. Three slanting agar tubes inoculated from the pleural fluid yielded each an abundant apparently pure growth of the organism used for infection.

CASE NO. IV.

The parts received for examination in this case were a piece of large intestine showing characteristic swine-fever ulcers, and both lungs. A large part of the right lung was hepatised; the hepatised area was moderately firm, and on section it showed marked swelling of the interlobular septa. The parenchyma was mottled with a lighter and a darker shade of red. A cover-glass preparation made from the lesion showed great numbers of short ovoid bacteria. The left lung was normal, and a cover-glass preparation made from its parenchyma showed no bacteria. The pig had been found dead on the morning of the day on which the parts had been brought to the laboratory.

Inoculated a rabbit (A) on the right side of the abdominal wall with a scraping from the lung lesion suspended in bouillon.

The rabbit was found dead on the fifth day after inoculation. Around the seat of inoculation there was an inflammatory swelling as large as the palm of the hand; on section this showed inflammatory congestion, œdema, and blanching of the tissues from necrosis. A cover-glass preparation from the blood of the ear showed no free bacteria, but many violet granules in the leucocytes (with methyl-blue staining). Slanting agar tubes inoculated from the heart blood yielded an abundant growth of flat, circular, transparent colonies. A bouillon tube inoculated with a loopful of blood from the heart was slightly turbid after twenty-four hours' incubation at 37° C., and subsequently became almost clear, depositing a somewhat ropy deposit at the bottom of the tube. On microscopic examination these cultures were found to be composed of non-motile bacteria of the fowl cholera type. As regards its mode of growth on the surface of agar and in gelatine-agar it was not distinguishable from the organism isolated from the three preceding cases. It formed no visible growth on potato.

The hepatised part in the right lung of the before-mentioned pig after having been immersed for half-a-minute in boiling water,[1] was cut into small pieces with a sterile knife, and given mixed with food to three adult sows (A, B, and C).

One day after the experiment was begun, sow A farrowed eleven pigs. On the twelfth day of the experiment this sow, which had hitherto appeared to be quite well, refused her food; on the following day she appeared to be very weak, and food was still refused. On the fourteenth day of the experiment the sow took some food and appeared to be rather better. The improvement was continued during the next three days, but on the twentieth day the animal was found to be very ill, and death took place during the evening. The following are the notes of the *post-mortem* examination, which was made fifteen minutes after death.

The right lung contains near its base a hepatised area about the size of a hen's egg. On section a turbid juice exudes from this. The cut surface is partly pink flesh-coloured (this is the firmest portion), and the rest is greyish pink. The cut surface of the firm portion is distinctly granular, and at one place hæmorrhagic in appearance. At one end the solid part shades gradually into the surrounding lung, but elsewhere it is well defined. A cover-glass preparation shows very numerous short bacteria. Left lung normal. Bronchial glands normal. Mesenteric glands normal in size; some of them congested. The ileo-cæcal projection is swollen, livid in colour, and œdematous, and on it there are several spots of necrosis. The large bowel contains no food materials, but about 6 oz. of blood, partly coagulated. The colon is intensely inflamed throughout, being generally of a livid colour, with here and there dark spots of extravasation. Jejunum much congested; no diphtheritic deposit and no ulceration. Stomach moderately filled with food materials. The cuticular portion has some adherent yellowish friable material. The fundus of the stomach is deeply inflamed. Spleen, liver, kidneys, and uterus normal. The right tonsil contains a dark-coloured hæmorrhagic-looking area about as big as a pea.

Slanting agar tubes inoculated from the lung lesion yielded an abundant growth of an organism which grew in the form of large, glistening, opaque white colonies, and a few colonies with a similar appearance were obtained on slanting agar tubes inoculated from the mesenteric glands. In both cases the colonies were found to be composed of a short motile bacterium, easily distinguished by its mode of growth from the swine-fever bacillus and from the organism cultivated from rabbit A. It coagulated milk, and formed a white growth on potato. No inoculation experiments have been made with this organism.

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[1] To destroy any swine-fever bacilli accidentally present on the surface.

Sows B and C were killed on the twenty-first day after they had been fed with the diseased lung. In the interval they had appeared to be quite well, and the *post-mortem* examination did not reveal any lesion in the organs of the thorax or abdomen.

Detached a slanting agar culture, which was the second generation from the heart blood of the before-mentioned rabbit (A), into about 10 cc. of a bouillon culture, which was also the second generation from the same source. Injected ½ cc. of the mixed cultures under the skin of the abdominal wall of a rabbit (B), and the same quantity into the pectoral muscle of a pigeon.

The rabbit was found dead on the eleventh day after inoculation. Around the seat of inoculation, and occupying nearly the whole of the right half of the abdominal wall, the subcutaneous and deeper textures were much swollen, and dissection showed that at this point the tissues of the abdominal wall were largely necrotic. A cover-glass preparation made from the blood of the ear showed no bacteria, and tubes inoculated from the heart blood remained sterile. An agar tube inoculated from the lesion of the abdominal wall yielded an abundant apparently pure growth of flat transparent colonies. A bouillon tube inoculated from the same source also yielded an apparently pure growth.

The above pigeon was alive and apparently healthy two months after inoculation.

Inoculated a rabbit (C) subcutaneously in the right groin with 5 cc. of a bouillon culture holding in suspension a culture on slanting agar; both cultures were the first generation from the abdominal wall of rabbit B.

The rabbit was found dead on the third day after inoculation. Around the seat of inoculation there was much inflammatory congestion and some hæmorrhage. No visceral lesions. Slanting agar tubes inoculated from the heart and from the local lesion yielded abundant apparently pure cultures.

Skinned and decapitated the above rabbit and then cut it into pieces, and gave the whole, together with the viscera, to a pig (A) about six months old. The whole was greedily consumed.

The pig was killed on the seventh day of the experiment; during the interval it had not shown any symptom of ill health. Throughout the whole of the small intestine the mucous membrane was more or less congested, and there was also some congestion of the gastric mucous membrane. The large intestine was normal throughout. The other abdominal organs and those of the thorax, as well as the tongue and throat, were normal. Of two slanting agar tubes inoculated from a mesenteric gland, one remained sterile, and the other developed two large opaque white colonies.

Took a culture on slanting agar, the second generation from rabbit C; poured into the tube about 6 cc. of sterile water, and detached the surface growth into this by the aid of a platinum needle. Injected

1½ cc. of this liquid under the skin of the right thigh of a pig (B), and 4 cc. of the same liquid through the left chest wall of another pig (both about six months old).

Pig B was killed on the thirteenth day of the experiment. At the seat of inoculation in the right thigh there was a pea-sized induration of the subcutaneous tissue. Several of the mesenteric glands were found to contain tuberculous lesions, and there was a small area of collapse near the edge of the left lung. All the other viscera were normal. Two slanting agar tubes inoculated from the centre of the nodule at the seat of inoculation remained sterile.

Pig C (inoculated through the chest wall) was also killed on the thirteenth day of the experiment. The *post-mortem* examination showed an adhesion between the parietal pleura and the left lung opposite the eighth and ninth ribs; at the point of adhesion there was a solid area of lung nearly as large as a walnut, and on section this was found to contain centrally a piece of necrotic tissue. Several mesenteric glands were enlarged and tuberculous. The organism was recovered in apparently pure culture from the necrotic lung tissue, but a slanting agar tube inoculated from the pleural lesion remained sterile.

CASE NO. V.

In this instance only the lungs were sent for examination. At the base of the left lung, and constituting about one-fourth of the organ, there was an area of hepatisation; on section the ground colour was greyish-pink, with hæmorrhagic-looking specks and streaks. The bronchi in this part contained a turbid mucus. A cover-glass preparation from the hepatised part showed an immense number of short bacteria, while a similar preparation from the normal lung tissue showed only an occasional organism (the lung was not quite fresh).

Slanting agar tubes inoculated from the depth of the lung lesion yielded, after twenty-four hours' incubation at 37° C., an apparently pure growth composed of small, circular, flat, transparent colonies, which, on microscopic examination, were found to be composed of short bacteria of the fowl cholera type.

Two rabbits (A and B) were subcutaneously inoculated on the abdomen with a scraping from the hepatised part.

One of the rabbits (A) was found dead on the third day after inoculation. A cover-glass preparation made from the blood of the ear showed only a few short bacteria. Around the seat of inoculation on the abdominal wall there was a lesion about 2½ inches in diameter; the textures of the abdominal wall were here infiltrated and partly necrotic. The peritoneum was inflamed, with intense injection of the vessels, particularly of the large bowel. Other organs normal. An agar tube inoculated from the blood of the heart yielded a culture similar to that obtained from the original lesion in the lung; a bouillon tube inoculated from the same source remained almost clear, while a growth formed at the bottom of the liquid.

The second rabbit (B) was also found dead on the third day after inoculation. The result of the *post-mortem* examination of it was almost identical with the foregoing, that is to say, very few bacteria were discoverable in the blood, and there was a comparatively large lesion at the seat of inoculation, and peritonitis. The organism was recovered on agar and in bouillon in apparently pure culture from the blood of the heart.

The organism cultivated from the hepatised area in the above-mentioned pig's lung, and from the blood of the two rabbits inoculated from that lesion, is morphologically identical with the organisms isolated from the preceding four pigs, and it also resembles these in respect of its mode of growth in artificial media.

A rabbit (C) was inoculated subcutaneously on the abdomen with 1 cc. of a bouillon culture, which was the second generation from the above-mentioned pig's lung.

The rabbit was found dead on the fourth day after inoculation. A cover-glass preparation from the ear showed only a few short bacteria. Around the seat of inoculation there was a lesion 3 inches in diameter. The peritoneum was inflamed, with coarse coagula of fibrinous lymph between the intestines. The organism was recovered in apparently pure culture from the heart blood and the lesion in the abdominal wall.

A rabbit (D) was subcutaneously inoculated on the abdomen with 1½ cc. of bouillon culture, which was the third generation from the before-mentioned pig's lung; this formed part of a flask of 40 cc. which had been incubated for twelve days at 37° C. The remainder of the flask (38½ cc.) was mixed with food and given to a six months' old pig (A). The whole was consumed.

The pig was killed on the seventh day of the experiment; in the interval it had appeared to be quite well, except for slight diarrhœa on the second day of the experiment. The *post-mortem* examination revealed no lesions in any part of the body, and two agar tubes inoculated from the mesenteric glands remained sterile.

In the above-mentioned rabbit (D) a swelling formed slowly at the seat of inoculation, and at the end of the third week it had attained the size of a hen's egg; after that it gradually declined. The general health of the rabbit did not appear to be seriously affected. The rabbit was killed forty-nine days after inoculation. It then had at the seat of inoculation a nodule about the size of a horse bean; centrally this was found to contain thick pus, and a cover-glass preparation made from that showed fairly numerous short bacteria, which were recovered in pure culture on slanting agar.

Took a culture on slanting agar, which was the seventh generation from the lesion in the original pig's lung, and detached it into about 6 cc. of sterile water. The culture had been incubated for five days at 37° C. Injected 1½ cc. of this liquid under the skin of the right thigh of a pig (B), and 4 cc. through the left chest wall of another pig (C); both pigs were about six months old.

Pig B was killed on the thirteenth day of the experiment; in the interval it had not shown any symptom of ill health. The seat of inoculation in the right thigh was not discoverable, even by dissection, and no lesion was discovered in any of the internal organs.

Pig C (inoculated through the chest wall) was also killed on the thirteenth day of the experiment. The seat of inoculation was not apparent in the tissues of the chest wall, or by any adhesion between the lung and the parietal pleura. Near the seat of inoculation the left lung contained, immediately under the pleura, a nodule about half the size of a pea; the centre of this was firm, dry, and yellowish-white in colour. Otherwise both lungs were normal, and no lesion was discovered in any of the other organs. A slanting agar tube inoculated from the centre of the lung lesion remained sterile.

(*To be continued.*)

PNEUMONIA AND PLEURISY IN THE HORSE.

By Veterinary-Major F. SMITH, F.R.C.V.S., Army Remount Department, Woolwich.

REGIONAL ANATOMY OF THE LUNGS.

AN accurate conception of the regional anatomy of the chest will be found of considerable service in diagnosis. On this subject I propose to say a few words.

It is usual to divide the chest of the horse into three regions, superior, middle, and inferior, and this division will clinically be found most useful.

The extent of the chest is considerable; a large region lies concealed under the fore legs, a part we may term the scapular region; while posterior to this a considerable surface is presented by what may be termed the available region. This term if adopted would simply indicate that portion of the chest which can be subjected to examination, it being obvious that the scapular region is not so available.

In Fig. 1 (p. 14) the extent and area of the chest is shown with a considerable degree of accuracy. I have termed it a diagram because my method failed to obtain a completely accurate representation of the chest for the purpose of this communication. But in spite of this it is prepared from photographs which were specially taken, and it may be accepted as quite accurate for all clinical purposes.

The points I wish to bring out in the figure are the extent of the scapular region B, D, E, C, and the considerable area of the available chest A, B, C, F. The line A F represents the last rib. B C is a line running parallel with the posterior edge of the triceps muscle; A B is one parallel to the lower edge of the longissimus dorsi muscle; F C is a line running along the costal ridge, but the lungs do not reach to within a hand-breadth of the bottom of the false ribs.

In the scapular region D is the head of the first rib and E its sternal insertion; the line B D runs close to the bodies of the dorsal vertebræ, while C E cuts through the cartilages of the sternum.

Anteriorly the cavity of the chest is narrow from side to side and from top to bottom, while in the available region of the chest it is both wide and deep. The lungs are higher in the body at A than they are at D, they are lower at C than they are at E.

As the chest is wider transversely, say through the sixteenth rib, than it is through the sixth, a greater bulk of lung should be situated

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from normal murmur; as a rule it is sharply defined, and this fact is an aid to diagnosis.

Harsh Respiration.—Harsh respiration is one in which the inspiratory and expiratory murmurs are each equally loud and distinct. I have occasionally heard it in the upper zone of the horse's lung, and have found it where the part has been pneumonic and consolidated, but also when the spongy nature of the lung has not been seriously impaired. The explanation of its appearance in the latter case is not very clear; it is possible that an error of observation has been made, and that an increased distinctness of the inspiratory murmur from lung consolidation below has led to it. In its typical form there is an absence of vesicular murmur, so that the sound must be produced in the bronchial tubes, or more correctly transmitted to the bronchial tubes from the glottis. This will be explained presently.

Tubular Respiration.—In bronchial respiration, or tubular breathing, we have one of the most characteristic lung sounds heard in the horse's chest during pneumonia. It has been described by Skoda as being capable of reproduction artificially, by placing the lips in the position of saying *ch* as in *ch*oir, and then drawing the breath in and out of the mouth. In the horse it is almost always an inspiratory and not an expiratory sound; occasionally it may be heard during both inspiration and expiration. The sound varies in pitch; that produced as described above would be a low-pitched blowing sound, as if the air were passing through a wide tube; but the converse may obtain, viz., a high-pitched note, as if the air were passing through a narrow tube, and this sound can be well imitated by placing the lips in the position to say *ch* as in *ch*urch, and drawing the air in and out of the mouth through the closed teeth.

Bronchial, tubular, or blowing respiration is only heard when the lung is consolidated, swollen, and immovable, at a time in fact when no air whatever is passing into it, and the important question therefore arises as to the cause of the sound. Chauveau has settled this by an interesting experiment on a horse, the subject of pneumonia and bronchial breathing. He has proved that the sound is produced in the glottis, and is merely transmitted to the lungs through the bronchi. This is no doubt true, but the fact remains that a lung may be extensively hepatised, and no trace of bronchial breathing heard; further, it will generally be found that when this form of breath sound is heard, it is confined to that portion of the lung above and slightly behind the point of the ulna, and is rarely heard elsewhere.

I am not prepared to offer any explanation either of the peculiarity in the region of bronchial breathing, or of the undoubted fact that of two lungs apparently, so far as the naked-eye appearance goes, in an identical condition as regards consolidation, the one during life yielded bronchial breathing, while in the other there was an entire absence of sound.

In dealing with pneumonia I will dwell at some little length on the significance of bronchial breathing and its importance.

Cavernous and Amphoric Respiration.—Cavernous respiration, and in its exaggerated form amphoric breathing, are the names given to a peculiar hollow sound heard during inspiration and expiration, and associated with cavities in the lungs. I have never heard it in the horse, though abscess cavities in the lungs are unfortunately only too common.

Râles.—When the air passing into or out of the lung can only find its way with difficulty, the obstructing agent produces a râle, ronchus, or rattle. The nature of the obstructing agent may vary; it is commonly an excessive mucous secretion from the tubes, which lodging there offers an impediment to the entry and exit of air; it may be pus formed in a cavity of the lung, producing a bubbling sound as the air passes through it into the cavity, or it may be an exudate in the alveoli of the lungs producing a rattle when the air sacs are distended; less commonly it may be due to a reduction or narrowing in the size of the tubes, such as would be produced by asthma, or by bronchial thickening.

It is obvious that the kind of râle produced in all these cases is not identical, and this has led to a classification of the sounds, such as *sonorous* or snoring, *sibilant* or whistling, *crepitant* or dry crackling, *sub-crepitant* or moist crackling, *gurgling* or cavernous râles, etc., but this by no means exhausts the varieties of râle which may be met with, and as in the majority of cases the production of each note depends upon some very slight difference in the character of the obstructing agent, it is readily conceivable that the kind of râle heard within the chest may extend over a very wide range.

Those most commonly heard are bubblings, both fine and coarse; squeakings, cooings, and occasionally clickings; less commonly rough, almost pig-like, grunting râles may be heard, and very fine crackling.

The significance of a râle will vary within wide limits. If the same sound can be heard over a wide area it is probably due to some mucoid obstruction in the larger bronchi; if coughing temporarily or permanently dislodges it, it is certain to be merely a bronchial secretion, or the breaking up of an exudate in the air cells of the lungs, such as occurs during the period of convalescence following pneumonia. If it is small in area, persistent, and always occupying the same region of the lung, it must be looked upon with suspicion, and in conjunction with other bad symptoms is almost sure to indicate abscess of the lung. If the râles are very fine, and are heard during ordinary, or on deep inspiration, they are significant of pneumonia.

Such râles partake of the character of crepitations or cracklings, but a lung on *deep inspiration* may yield crepitant râles without being affected with pneumonia, though such a case must be regarded with grave suspicion if any constitutional symptoms be present. The most perfect crepitations may be heard while artificially distending healthy lungs removed from the chest.

It can thus be seen that with one or two exceptions a râle teaches us nothing of a distinctively diagnostic nature; the most alarming sounds proceeding from the tubes may be entirely, and for a time permanently, displaced the next time the animal coughs, and therefore are probably not of serious importance. The permanence of the sound, its limited area, and its unvarying position, on the other hand, are signs of the greatest, and may be gravest, import. Excluding, therefore, from the following statement the crepitations of early pneumonia, we may say that the significance of any râle is in direct relation to the progress of the case; if the patient is doing well the râles need not alarm us, if doing badly they tend to increase our anxiety.

Bronchophony is the term employed by human physicians to indicate the fact that the voice of the patient can be heard with undue distinctness through the chest wall, and at first sight it would appear to be an aid to diagnosis not available in the horse. But this is not correct; I shall prove that it is both available and of great utility.

It is quite true we are unable to get the animal to produce a laryngeal sound at will, but we can utilise for the purpose of bronchophony the grunting sound so frequently present in lung trouble, or if this is absent we can artificially produce a sound in the trachea, which for all intents and purposes of diagnosis may be regarded as a voice sound produced by the animal itself.

The mechanism of bronchophony, like that of bronchial breathing, is that it is a laryngeal sound transmitted down the trachea to the larger bronchi; like bronchial breathing it indicates lung consolidation, but whereas bronchial breathing is an inspiratory act, bronchophony is purely expiratory. The two may be associated, or one may exist without the other.

When the voice sound is produced by the patient grunting, the grunt may be heard over the chest wall as if it proceeded from immediately beneath the stethoscope. This is the characteristic feature of the sound; it appears to be produced in the portion of lung being examined, but of course agrees in time and sequence with the grunting, loud or suppressed, heard at the nostrils.

Like bronchial breathing it is not heard all over the lung, but is usually most distinct over the same region as bronchial respiration, namely, above and behind the point of the ulna. Bronchophony once heard can never be forgotten; it indicates lung consolidation, though it is often absent even when the lungs are quite solid.

If grunting is absent we may yet test the voice sound by tapping lightly on the exposed surface of the trachea high up in the neck. The tapping can be heard with the utmost distinctness, as if proceeding from under the instrument on the chest wall; to distinguish this form of bronchophony we may term it the "tracheal reflex."

Unfortunately, as I have previously mentioned, bronchophony and the allied sounds are often absent even when the lungs are quite firm and solid, and I can offer no explanation of their absence, though it is generally believed to be due to obstruction of the bronchi. On *post-mortem* examination there is nothing to distinguish the firm lung which transmitted bronchophony from the firm lung which did not.

Pectoriloquy.—A term employed by human physicians to indicate that the voice sound passes through the wall of the chest and up through the stethoscope to the ear of the observer. The difference between it and bronchophony is, that in the latter the sound seems to pass away from under the instrument, while in the former it appears to pass *up* the instrument with the most remarkable distinctness, so that if the person whispers each syllable is clearly heard. The only approach to pectoriloquy in the horse is the sound heard in swallowing, and that is transmitted so clearly and sharply that it is almost impossible to believe it is being produced a few feet away. In human practice pectoriloquy is almost diagnostic of a cavity in the lung, but in veterinary practice this is not so, for the sound may frequently be heard in simple lung consolidation without abscess, and usually over the region of bronchophony.

The *Pleural Sounds* are generally described as frictional, such as may be produced by rubbing two pieces of leather together, but this by no means fully describes their character; they are often distinctly *creaking*, indeed more often than not, and are heard not only at inspiration, as some believe, but just as markedly at expiration. Further, the rhythm of the creaking does not necessarily agree with the rhythm of the respiration, that is to say, the creaking may be continuous so that one cannot judge by listening to it when the pause in the breathing occurs. This is so unlike what is generally taught, and yet so diagnostic of the condition, that we must stay for a moment to enquire into the cause.

During an ordinary inspiration in the normal chest the lung glides frictionless over the ribs both at inspiration and expiration, but when the pleural surfaces become inflamed they are dry and rough, the lung slides over them with difficulty, even with great difficulty, and it momentarily catches here and there, and then goes on, and again catches, so that the act of inspiration or of expiration is at an end before the lung has had time to attain its full state of distension or of collapse, the result being the pleural surfaces are continually on the move.

Pleuritic friction is commonly heard at the lower part of the chest, behind and above the elbow, in fact at the usual seat of pneumonia. It will be found on *post-mortem* examination that the pericardium rarely escapes being drawn into the inflammatory attack, and during the early stages of pleurisy a well-marked friction sound synchronous with the heart's action may be heard over the point of the left ulna. One would think that the sound would be best heard further forward, immediately over the heart, but this is not so. In the latter position pericardial friction may be absent, and the heart sounds distinctly heard, while in the former position each beat of the heart is accompanied by a remarkably clear friction sound, completely annulling the heart sounds. I am rather at a loss to explain this fact.

Pleuritic friction never lasts more than a day or two, and often only a few hours. Early examination of the chest must therefore always be made.

Auscultation of the chest can only be satisfactorily practised when the surroundings are absolutely quiet, for even the noise of a horse feeding is quite sufficient by disturbing the observer's attention to cause a sound to be lost. Whenever possible I do my auscultating at night, so as to ensure quietude, and this is largely contributed to by using sawdust as a bedding; on this both attendant and horse may move without disturbing the examiner.

I have previously spoken of the difficulty presented to the auscultator by naturally dull chests. The next difficulty he has to deal with is the intestinal murmur. These invariably appear to be immediately under the instrument when sounding the inferior region of the chest; at times they are so persistent that they completely mask the lung sounds, and often at the most critical period of one's examination. They are particularly active just after causing the horse to take a deep breath, and often necessitate this process being repeated. Only time and patience can overcome the difficulty created by their presence.

PERCUSSION.

When the lungs are in a normal condition and the chest wall is tapped with the fingers, a clear note, known as pulmonary resonance, is obtained.

The degree of resonance depends upon the thickness of the chest wall, and the character of the underlying structures. For example, in the inferior third of the chest it is nothing like so clear as in the middle third, owing to the extra amount of muscular tissue and the cartilages of the ribs. The resonance increases from below upwards until the upper third of the chest is entered, when it again decreases in intensity owing to the muscles of the back. Further, the resonance decreases from front to rear, due to the existence of certain structures filling in the concave posterior face of the lungs. In fact it may be said that where a great thickness of material covers the ribs the sounds are dull, and where the chest wall is lightly covered and thin the sounds are clear. The fat which so plentifully covers the ribs cf dealers' horses almost prohibits percussion.

In my hands percussion has not proved so useful or so accurate as it appears to have done in the hands of others. I can gain from it a fair notion of the extent of hepatisation of a lung in the lower region of the chest, and this is useful in corroborating the diagnosis obtained by auscultation. Further, it is valuable in indicating the presence of fluid in the chest, and more particularly the rate of its production; but I cannot map out an organ with precision by means of percussion, nor diagnose an abscess, and in the upper zone of the lung a moderately clear sound by no means indicates that the part is free from hepatisation.

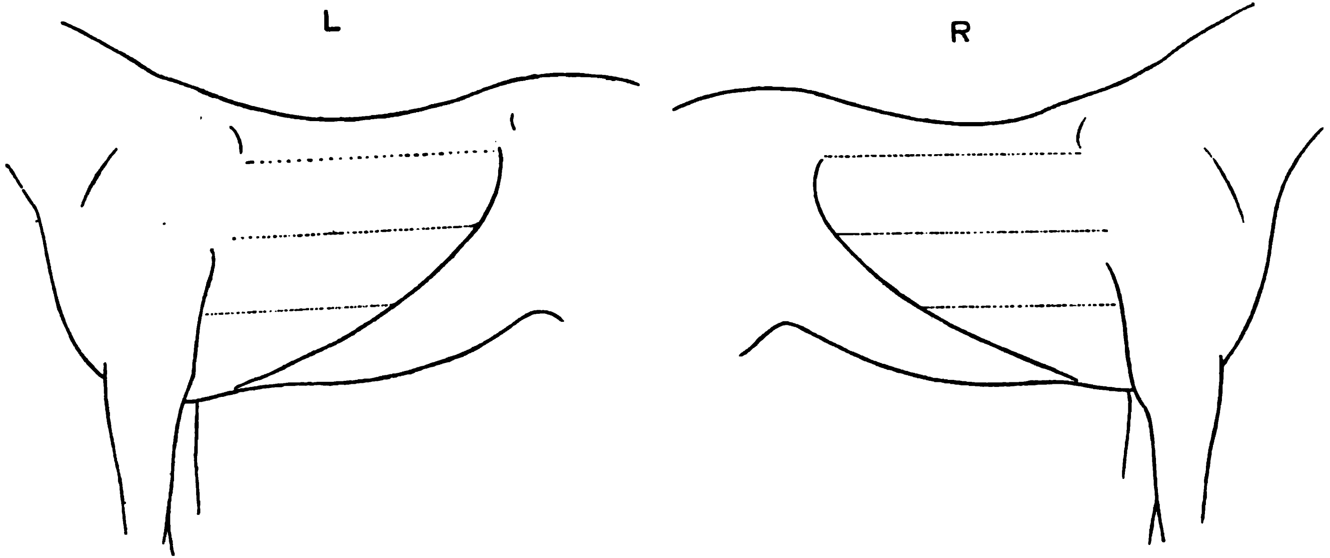
An explanation of this paradox is simple. When a lung is hepatised no air is entering it; it is swollen and heavy. In the lower region it lies on the sternum motionless, and the full effect of this dead weight is readily determined by the absolute dulness on percussion. But in the upper region of the chest the hepatised area cannot be readily determined by percussion, and quite a clear note can at times be obtained from a region where not a trace of air is entering!

We must therefore be careful in drawing conclusions from percussion in the upper region.

Dulness on percussion will vary from a slight diminution of the normal resonance to absolute dulness of a doughy character. The former is present in commencing pneumonia, the latter in complete consolidation, or in effusion into the pleural sac.

There are certain percussion notes heard in the human chest when the lung contains cavities. I have heard nothing like them in the horse, but as the sounds can be reproduced artificially with considerable accuracy, I mention them here that others may look for them. *Amphoric percussion* can be imitated by tapping the cheek with the mouth slightly open, the cheek being drawn tensely over the teeth. *Cracked pot sound*, also significant of cavity, resembles the noise produced by placing the hands in apposition with the palms hollow, and striking them on the knee.

In percussing the chest the fingers of one hand are laid flat on the part and struck by the tips of the fingers of the opposite hand; or, *Name or Number*.. *Date*................................ *Hour*......................



Remarks.

FIG. 2.—A chest chart.

the case. If, as in army practice, horses are reported or brought to the notice of the veterinary surgeon immediately they show signs of being out of health, a distinct period may often be observed to elapse before active inflammatory chest symptoms appear. I say often, because this condition is not invariable, but it is so frequent that I never consider any case of high temperature with rapid pulse and breathing safe until the third day, and experience shows that in some cases even a longer period than this may elapse before invasion.

This particularly applies to pneumonia; it is less evident, though present, in pleuro-pneumonia and pleurisy. I shall, however, for the present confine my remarks to pneumonia.

PNEUMONIA.

Invasion Period.—Of the forty-seven cases of pneumonia which I record in my tabular statement thirteen were found affected on admission, nine were attacked on the first day, seven one-and-a-half days after admission, seven two days after, five three days, three four days, one six days, one ten or eleven days, and two uncertain.

We may say, speaking roughly, that on admission a little more than one-third yielded physical symptoms of pneumonia; one-half of the cases had declared themselves by the end of the first day, and more than two-thirds had given undoubted evidence of their trouble by the end of the second day after admission.

I never feel satisfied with a doubtful case until the third day has passed, when the chances of infection become considerably reduced. This leads to the very important rule of a careful examination of a suspected chest twice a day; there are many suspected chests which I have auscultated three times in twelve hours.

I am quite aware of the impracticability of doing this in general practice, and only record here what should be done if opportunity and time permit. One of the most common conditions with which I am acquainted in chest trouble is to find both lungs perfectly clear, say at the morning examination, while by the evening they are giving undoubted evidence of change. By establishing a system of frequent examination valuable time in the treatment of a case may be saved, while we are spared the unpleasantness of having an unsuspected case of pneumonia brought home to us.

The symptoms which these as yet undeclared pneumonia cases exhibit do not differ essentially from those shown by a subject of the disease, save and except that the chest is clear, and air entering all over it. There is fever, rapid pulse, hurried breathing, but at present beyond the fact that fever exists we are absolutely unable to go. My table previously quoted shows that this state may last three days, or even longer, but if the lungs have escaped the third day the chances of an attack of pneumonia become considerably reduced.

The Urine a Guide.—During the doubtful period our suspicions of an impending attack of pneumonia may be greatly strengthened by an examination of the urine, and I use the word strengthened advisedly, for on this point I am unable to make a definite and positive statement, nor will it be possible for some time to come to settle the point. This, however, is what I have observed. The horse the subject of an impending attack of pneumonia, in which no symptoms of the disease can at present be detected by a physical examination of the chest, will in all probability be found to have proteoses in its urine. These proteoses are common in chest inflammation, but they often appear in the urine before the lungs are observably affected. I should, however, state at once that the same may be found in pleurisy and pleuro-pneumonia, so that the only warning the proteoses are capable of affording, so far as I can see at present, is to guard us against a hasty prognosis, and strengthen our opinion as to impending lung trouble.

I have endeavoured to speak with caution as to this symptom, for a sufficiently prolonged series of observations may show that proteoses are not excreted exclusively in impending chest inflammation, nor in fact are they always present in this affection, for I have met two notable and important exceptions. Still, so far as I can at present see, the rule appears to hold good, and is well worth further and extended enquiry. To look for pneumonia in a test tube may not recommend itself to some minds, but I would have such remember that albuminuria, diabetes, and typhoid fever are amongst the diseases thus looked for or aided in diagnosis.

Single and Double Pneumonia.—Pneumonia may be single or double, viz., affecting one lung or both. According to my data, single and double pneumonia have appeared with equal frequency, nor can I observe any difference between the susceptibility of the two lungs; right and left pneumonia are equally common. It is not often, however, that a well-marked case of single pneumonia becomes double, though I have notes of several such cases.

We have indicated that a case of impending pneumonia yields on auscultation a perfectly clear chest, that is, air is entering freely and normally all over it, but such a state of affairs cannot last very long. Within a few hours of seizure, or, at any rate, some time within the third day, rarely beyond it, evidence is present that the lungs are undergoing some change. This evidence is furnished by the respiratory murmur in the lungs, which becomes of an oppressed character.

I have used the term "oppressed" to describe the premonitory pneumonia condition, as I can find no better. A murmur is still present in the lungs, though it is not the normal murmur with which we are acquainted, but one deficient in quality. Air is not entering the lung as freely as it should, yet a certain amount is finding its way in, and this gives rise to the oppressed murmur.

Sometimes when sounding a chest in the early stages of pneumonia, nothing can be heard but a confused humming sound which drowns any respiratory murmur that may exist. This humming noise is not produced by the lungs, but in the muscles covering the chest wall. When humming is best marked muscular rigors are generally if not invariably present, and when these cease the humming ends.

Where Pneumonia commences.—The limit of the oppressed murmur can be accurately defined on comparison with the healthy portion of the lung, for no fact in the whole range of pathology can be better established than that the whole of a lung or lungs is not attacked with inflammation at one and the same time. It may be stated as a rule, to which I have only met two or three well-marked exceptions, that pneumonia commences in the inferior and anterior portion of the lung; sometimes not more than one-sixth, but more often one-third of the inferior lobe of the lung is attacked. Whether the anterior lobes are affected at the same time as the lower portion I cannot say, owing to the difficulty and often impossibility of examining them through the thickness of the triceps muscle. It is, however, quite certain that the anterior lobes are commonly affected with inflammation, and it is seldom that one examines *post-mortem* a pneumonia in the lower third of the lung without finding the anterior lobe of that side also implicated.

It is therefore to the lower third of the chest, behind or inside the point of the ulna, that the most careful examination of the lung must be made. If this part is clear the chances are enormously in favour of the whole lung being free from disease, though an examination of the remainder of the chest should by no means be neglected.

Early Stage of Pneumonia.—Returning once more to the oppressed respiratory murmur, it is to be noted that, as a rule, it is quite free from any crepitations such as are regarded as indicative of pneumonia in the human subject, but in some favourable cases crepitations can be obtained by causing the patient to take a deep breath in the manner before described, and then at the end of inspiration a well-marked crackling sound may be heard. In a large number of cases no crackling is present, and personally I do not care whether it is present or not; the oppressed respiratory murmur is a much safer guide, and if this exists it is safe to say that pneumonia will follow.

Associated with the oppressed murmur found in the lower part of the chest, is often a well-marked increase in the sound of the normal respiratory murmur in the upper part of the lung. The mechanism of this condition is easy to understand—the upper part of the lung is admitting more air owing to the condition of the lower part.

In some cases of impending pneumonia even the oppressed murmur cannot be detected, in fact the chest refuses to yield any murmur; such cases will generally be found associated with those chests which, even when healthy, yield no respiratory murmur during ordinary breathing, and which have been dealt with in a previous section. The test of a deep inspiration will generally decide the matter; air is then discovered to be entering imperfectly over the troubled area, while over the normal part of the lung the natural respiratory sound is heard. More than one deep inspiration will be necessary in making this examination, for the murmur rapidly dies away.

The condition of lung yielding an oppressed murmur is probably that of engorgement, with a moderate exudation into the alveoli.

Consolidative Stage of Pneumonia.—The period during which an oppressed murmur is present is as a rule only a matter of a few hours; more or less quickly it is succeeded by an entire absence of sound, which even the deepest inspiration may fail to restore, though in some cases it may be relieved by a few ill-defined and distant "squeaks." Such symptoms will be yielded by a lung which is gorged with blood, firm, swollen until its pleural capsule is quite tense, while its alveoli are filled with inflammatory exudate. Indeed in very many cases we have made out all that is likely to be ascertained until either resolution sets in, or the symptoms indicate dissolution; for an entire absence of sound is also associated with a condition of lung rather more advanced than that just described, viz., where the lung is so firm as to cut and look like liver, and to which the term hepatisation has been so well applied.

While all this is occurring the disease is extending, at anyrate for the first few days; the extension is almost invariably in an upward direction, and as the lungs are longer along their upper than their lower edge, the extension is also backwards and possibly forwards.

This extension can be perfectly well mapped out from day to day until it comes to a standstill, the newly invaded portion of lung giving the oppressed murmur to be followed by an entire absence of sound.

In some cases of disease, but by no means in all, a peculiar kind of inspiratory murmur is present, occasionally as early as the second or third day of the attack; I allude to bronchial or tubular breathing. The character and mechanism of this sound have already been fully described.

The significance of the sound I cannot fully explain. In the human subject it is considered to indicate consolidation of lung tissue, the normal bronchial breathing being conveyed to the surface of the lung and thus to the chest wall. But this cannot be the entire explanation, for there are many cases of disease where in spite of solidification of lung substance there is not a trace of tubular breathing; again, as we shall later point out, it may at times be heard in hydrothorax even to the bottom of the chest, though the lungs are shrunk from hydrostatic pressure and not even touching the chest wall. In these cases, of course, the sound is transmitted by the fluid, but it is difficult to imagine a greater contrast than a large hard swollen lung resting against the chest wall and unable to conduct the bronchial breathing to the surface, and a shrunken collapsed lung, bathed in fluid, lying some distance from the wall, but which transmits the bronchial respiration in the clearest manner possible.

It is strange why bronchial breathing should so often be absent even when the lungs are solid, nor, as I showed just now, can it be regarded even when present as necessarily indicative of consolidation of lung. The sound is best heard in two places, over the main bronchus and behind and above the elbow; it may, of course, be heard in almost any part of the lung, but the two places named are the most common.

I mentioned previously that tubular breathing may be entirely absent in a case from beginning to end, and I am rather inclined to regard its absence as an unfavourable sign, for on reference to my notes I am struck to find that many horses exhibiting tubular breathing recovered, while several in which it was absent died. Tubular breathing persists as a rule for several days, in fact until convalescence sets in or death takes place. I have known it disappear with remarkable rapidity.

Besides the presence of tubular breathing, a pneumonic case may exhibit the peculiar condition known as bronchophony almost any time after the third day, though I have met with it earlier.

Bronchophony is indicative of lung consolidation, but unfortunately it is often absent, probably due to the fact that a patent condition of the bronchial tubes in the consolidated area is necessary for its production; when once heard the sound can never be forgotten.

Grunting during expiration is common in pneumonia and pleurisy; it may be present without a trace of pleuritic trouble, a fact which is probably against the experience of most practitioners. This grunting can be distinctly heard in some conditions of lung, and constitutes bronchophony. If the grunting is slight it gives rise to a blowing *expiratory* sound in the chest, readily mistaken for tubular breathing. Both the tracheal reflex and grunting bronchophony may be absent in consolidation of the lung, but it is rare that another sound escapes detection, namely swallowing, which can be heard as pectoriloquy.

When lungs are consolidated they conduct the heart sounds over a very large area of the chest. This symptom by itself must not be too hastily regarded as proof of consolidation, but it is useful to remember when considering the case in all its bearings.

As consolidation of the lung takes place the note on percussion becomes duller, and the rate at which this consolidation is extending may be indicated by clipping a mark on the chest wall from day to day. In the earlier stage of pneumonia, while still a little air is entering the lung, the percussion note is only slightly affected.

Let us clearly understand that all the physical signs we have been discussing indicate only two conditions, viz., the engorged and hepatised stages of pneumonia, and one follows the other as certainly as the night follows the day. The condition of hepatisation is that known as red, where the lung on section has the consistence and colour of liver. It is well for the patient if the disease process stops there; in the majority of cases placed under favourable conditions it will go no further, and the third and final stage of such cases is resolution or the clearing up of the effused products.

As this is the normal termination of the process we may fitly consider it here, and attempt to determine how long the case lasts before resolution occurs, and how the existence of the process is recognised.

Resolution.—A case of pneumonia which is going to terminate satisfactorily should show definite symptoms of improvement about the middle of the second week; many indeed if taken early will show this improvement in the first week, but in severe cases the second week is more common. In the table appended to this paper is a column showing the period of fever in days; I am always satisfied to observe a fall of temperature between the eighth and the eleventh day, and with it of course a reduction in the pulse and respirations.

In the human subject it is believed that the onset of favourable symptoms in pneumonia is sudden, in other words a *crisis* takes place. Within the space of a few hours the temperature suddenly drops, and with it the pulse and respirations. An examination of my charts does no doubt reveal in some of them what might be considered a crisis curve, but as a rule it may be stated that it requires about three days for the temperature to reach normal, and during that time it is progressively falling; there should not even be a rise in the evening temperature, but a steady fall of about one degree every twenty-four hours. This is termination by *lysis*. There is no temperature curve proper to pneumonia, neither during the fever period nor the decline, and many varieties of curve are given by cases of equal intensity.

The decline in the temperature and pulse should be followed by a corresponding fall in the respirations, and my experience leads me to regard with suspicion any case where the respirations do not undergo a marked decrease in number simultaneously with the temperature, though such cases are by no means always fatal.

With the fall in the temperature, pulse, and respirations, we may shortly expect some improvement in the chest sounds, but not uncommonly a few days will elapse before we can be certain of absorption taking place. In other cases the chest readily gives evidence of clearing up, and I take it that the difference here shown is a question of the intensity of the lung changes and the recuperative powers of the patient.

For some two or three days after convalescence is established the most careful auscultation of the chest will often reveal no change; there is an absolute absence of air sound, with marked dulness on percussion, but within the first week there are certain sounds accompanying a very modified or oppressed respiratory murmur, which indicate that air is commencing to find its way once more into the alveoli; these sounds are moist râles, cracklings, or squeaks, heard both at inspiration and expiration, and generally commencing at the border of the sound and affected lung. It is, I think, an undoubted fact that the lung clears up in an inverse direction to that of invasion; while, for example, it is invaded from below upwards, it clears up from above downwards. No positive statement can be made as to when absorption of the lung exudate commences to occur, or how long the process lasts. I have a note of five days having been sufficient to clear up a portion of lung, but this is very exceptional; many cases have been examined by me two, three, five, and six weeks after all acute symptoms have passed away, and the lungs were even then far from clear; in fact, I think there can be no doubt that some lungs never entirely clear up.

A nasal discharge during convalescence is frequent; it is a satisfactory condition, and indicates a removal of the pulmonary exudate.

Dissolution.—If a case of pneumonia shows no improvement during the second week the prognosis is very grave, and if the third week be entered upon with no reduction in the pulse rate and respirations a fatal result may be anticipated.

During this time the changes in the lung are simple enough, but the physical symptoms obtained on auscultation are not so evident. The lung changes are as follows: Areas of irregular outline in the red hepatised region, varying in size from a small nut to a clenched fist, turn grey in colour, and finally break down. I used to describe these grey areas in my note-book as grey hepatisation, but Professor M'Fadyean has pointed out to me that such areas in no way correspond to the so-called grey hepatisation of the human lung.

These grey areas precede necrosis of the lung substance, and if the patient lives long enough the grey material will become liquefied or grumous, and be cast off in the form of an abscess.

A lung in this condition presents a very characteristic appearance; the grey areas, of what may conveniently be termed grey degeneration, resemble on section a magnified nutmeg in the lung, and the contrast in colour between them and the hepatised tissue around is very marked. Surrounding the grey mass is a quite sharply defined grey border.

A later stage than this occurs when the grey degeneration is converted into a necrotic mass containing grumous cheesy material often extremely offensive. If a slice of the lung be placed under a tap the grumous grey offensive material is washed away, leaving behind a cavity, the wall of which is intensely discoloured; and if many such cavities exist, the appearance of the lung after washing can best be described as riddled with holes.

Sometimes, but rarely, there is one vast irregular, cheesy, discoloured, semi-liquid, necrotic mass in the substance of the lung, which when washed away leaves nothing in its interior but blood-vessels with greatly thickened walls, the lung substance having been destroyed ; the wall of such a vast sac is merely thickened pleura.

The rule, however, is that the cavities are relatively small and very numerous, though they frequently by coalescing form cavities of no inconsiderable size, this especially being the case in the anterior lobes of the lung. In fact, the most common seat of lung abscess is in the anterior lobes, though if a case has lived long enough through the attack hardly any part is free from them.

The cavities I have described are situated within the affected area of lung, but this is by no means their exclusive seat; they may be found in portions of lung which, judged from their external appearance are quite normal, and it is only by cutting into this normal-looking portion of lung that the abscess is found. This is especially the case in the superior and posterior part of the lung, and it is possible that these abscesses are secondary to those in the inflamed area.

It is only occasionally that true pus can be found in lung abscess. We have thus described two conditions. 1st. Red hepatisation, enclosing grey degeneration, within which are necrotic masses ; and 2nd. Lung abscess, without any hepatisation surrounding it. Besides these a third condition may be found, and that is circumscribed areas of congestion about the size of a half-a-crown, situated between and among healthy portions of lung. These islands of congestion may possibly be the commencement of secondary abscesses ; that is a question the pathologist must settle, but the *post-mortem* appearance is constant and characteristic.

Such are the changes occurring during the stage of dissolution in pneumonia ; let us consider how they may be recognised during life.

Any horse which has passed through two weeks of pneumonia without the disease yielding, may be considered to be on the road to necrosis of the lungs ; of course many will die from necrotic pneumonia before that period, especially, I think, the pneumonia affecting old horses. I have found the lungs of the latter necrosed in three or four days from the commencement of the attack, but in young horses the tissues apparently possess greater resistance ; with these lung necrosis generally commences towards the end of the second week or beginning of the third, and terminates life during the third week, sometimes the fourth, and in one case in my practice the fifth week.

Recognition of Lung Abscess.—During this time though the animal may feed, and the temperature and the pulse rate fall, the respirations will be found still markedly increased, or at any rate not less than those of the first and second week, and I regard, as previously mentioned, the respirations in such cases as being of almost as much value as the chest examination. If the patient has not previously given an expiratory grunt he will do so now ; some of them grunt loudly and persistently, others only grunt loudly while lying down, for it is not uncommon at this late stage for the patient to try and take rest to ease his tired and weak limbs. There is frequently a discharge from the nostrils which looks and often is, I think, distinctly purulent; this discharge towards the final termination may or may not be tinged with blood. In one of my cases actual hæmorrhage finally took place in such volume as to terminate life. The loss of condition, in spite of the appetite, is considerable, and the expression is anxious.

On auscultating such a chest we must not be surprised to find that areas previously dull are admitting air, and so marked is this that we may easily be thrown off our guard, and believe the lung to be clearing. But the murmur is not a normal respiratory one; it is of varying tone, sometimes harsh, sometimes muffled, and it is *heard as distinctly at expiration as at inspiration*, which is abnormal. Mixed up with it or not as the case may be, are liquid bubblings, cooings, clickings, and musical notes of various kinds; these may be heard both at inspiration and expiration, sometimes only at inspiration. They may disappear and re-appear, the former following a cough, which is low, painful, and without quality. Over certain areas, usually over the lower half of the lung, well marked tubular breathing and bronchophony can generally be detected. Over the upper part of the lung the murmur is at times harsh, at others obscure, though probably, so far as we knew, this portion of lung was previously unaffected by disease.

Over certain regions, often in the middle third of the lung, may be detected circumscribed areas where the liquid bubblings, creakings, cooings, and clickings are most intense, and heard both at inspiration and expiration; moreover, these have no tendency to temporarily disappear, such as those we previously described. In such areas actual abscess cavities exist.

Percussion Fallacy.—One of the most curious features in a chest affected with necrotic pneumonia is the remarkable change undergone by the percussion note. I have noticed repeatedly that a chest which for a week or two was absolutely dull on percussion half way up, was in a day transformed into one yielding almost a clear percussion note all over, and this even in places where *post-mortem* examination failed to reveal the presence of abscess, while the most marked evidence of consolidation existed!

Why should the percussion note change? I cannot offer an explanation for all the cases I have observed, but I have recently found that in such chests a leak in the pulmonary pleura has allowed air to escape through an abscess into the cavity of the chest. Whether this explanation will suffice for other observed cases I cannot say, but I place this rather remarkable feature on record for future observation and research. The sudden clearing of the percussion note would appear to be due to pneumo-thorax.

Respiration Fallacy.—Again, I am at a loss to explain the respiratory murmur being heard quite suddenly over an area which had previously been impervious to air, and in which, perhaps, not an abscess exists. That the sound is reflected I have no doubt, for I have noticed the same in the latter stages of effusion into the thorax, when there has been no lung opposite the chest wall. Of the existence of the phenomenon there is no doubt; that the murmur heard is not a true respiratory murmur, is, I think, equally certain, though it requires an educated ear to tell the difference between it and the normal murmur. All I can say of it is that it is coarser in character, and the *expiratory* *sound is as loud as the inspiratory*, but why a lung absolutely impervious to air for many days should suddenly transmit a murmur closely resembling a normal one, is quite beyond my comprehension.

Under any circumstances, neither the alteration in the percussion note, nor the appearance of respiratory murmurs in a previously solid lung, is likely to deceive the practitioner, for the patient is obviously on the eve of dissolution and beyond hope of recovery.

The abscesses found in necrotic pneumonia are always multiple ; a single abscess in the lung is not hopeless. I have found such in an otherwise absolutely healthy horse, caseated, and firmly encysted in the substance of the lung. Of its origin I knew nothing. A wax model of this specimen will be found in the Museum of the Royal College of Veterinary Surgeons.

It might appear from the foregoing that the diagnosis of abscess in the lung is a simple matter. This is not so, for as a matter of fact there will always be many more abscesses, even near the surface of the lungs, than could be localised during life, while of those in the deep-seated parts of the lung, towards the central line of the chest and in the anterior lobes, we shall absolutely know nothing.

Deep-seated Pneumonia.—This seems a convenient opportunity for speaking of deep-seated pneumonia. Such will be found in most *post-mortem* examinations, the favourite place being towards the middle line, where the inner face of the lungs are in apposition. Whether this position is primary or secondary in origin I do not know, that is to say, whether this portion of the lung is ever implicated at the commencement of an attack I cannot say, but I see no reason why it should not. It is certain we can know nothing of it until it is revealed by *post-mortem* examination.

Pneumonia of the anterior lobes may also for clinical purposes be regarded as deep-seated, inasmuch as it is practically inaccessible. In spite of this an attempt should always be made, by advancing the leg, and pushing the disc of the phonendoscope forward under the triceps near the ulna, to make an examination of the anterior lobes. In favourable cases a murmur can be heard through the triceps. The existence of a normal murmur in the scapular region is satisfactory, but its absence by no means denotes disease. See *ante.*

I feel certain there are cases where the lungs are affected in the anterior lobes, while the available region of the chest indicates a freedom from pneumonia. Gradually as the process extends backwards the inferior border of the lung posterior to the heart becomes affected, and the disease is then capable of recognition. Perhaps, in fact, this may in some cases be an explanation of the latent or incubative period of pneumonia shown in my table. Whether it is or is not may be settled in the future ; in the meantime the practical fact remains, and it forces once more on our attention the rule never to definitely pronounce lungs as free from pneumonia until at least three days have elapsed from the primary onset of fever.

Hunting's Views on Necrosis of the Lungs.—Returning once more to necrotic pneumonia, it is necessary to draw attention to an able communication by Mr W. Hunting[1] on "Putrid Pneumonia in Horses," in which he states that the grey degeneration which precedes necrosis requires three months for its formation, that when it is present in the lungs it is evidence of a previous attack of pneumonia, and further, that in no case of recent pneumonia has he met with this grey change.

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[1] Veterinary Record, 18th January, 1890.

The first statement is contrary to my experience; I am sure grey degeneration can occur in two weeks, and I suspect in even a much shorter time than this. This expression of opinion in no way affects the correctness of the observations made by Mr Hunting, but it does affect the conclusions drawn from them.

Fœtid Breath not a Necessary Accompaniment of Necrosis.—The fœtid breath which accompanies what Mr Hunting terms putrid pneumonia I have met with, but the lungs may be necrotic and fœtor of breath absent; this I have repeatedly observed in young horses, even though the lungs were riddled with abscesses. I think this point of some importance, for if we wait for a putrefactive odour to decide in acute pneumonia of young horses whether the lungs are broken down or not, we will generally have to wait until after death, at which time an undoubtedly fœtid odour can be detected on opening the chest. On the other hand, the well-named putrid pneumonia of older horses does yield an odour readily detectable on entering the sick box, and I suggest, with my imperfect knowledge of the latter disease, that the two conditions may not be quite identical. They are certainly not in point of time, for the pneumonia which in old horses is followed by fœtid breath is in my limited experience only a matter of a few days' duration.

It is conceivable that some of the cases of putrid pneumonia are caused by plugging of the blood-vessels of the lungs. In death from ordinary pneumonia one is struck by the firmness of the clots existing in the vessels.

We have at some length followed out the course of pneumonia in the horse, the changes it produces in the chest, and the physical signs by which we may gain an approximate idea of what is going on within. We must now consider the temperature, pulse, and respirations.

Temperature.—I have previously mentioned that no typical curve can be obtained from pneumonia. Of the very large number of charts in my possession, a certain proportion of them give a somewhat similar curve, but this proves nothing, and is what we might expect. As a rule the highest temperatures are registered in the first week, and more commonly on the first, second, and third day of the attack than any other. It is generally between 104° and 105° on invasion. The curve I like best is one with few steep gradients, indicating that the difference between the morning and evening temperature is not too great, and finally coming steadily down so that at each observation the temperature is less than the preceding record.

I have two charts showing an enormous fall in temperature on the second day, in fact from 106° to 100°, with a rise a few hours later; one of these cases was fatal. In another fatal case where the gradients were steep and the curve irregular, 100° was touched once during the second week, yet the case terminated fatally. I mention these to show that a sudden return to normal temperature must not cause us to slacken our vigilance.

The daily temperature is an excellent guide to the progress of the case, but I have previously given reasons for believing that in third week cases the respirations are even more useful.

It may often be observed that after the temperature has reached normal slight rises occur for a day or two. The small rises are unimportant, but any big rise must be regarded with suspicion and the case carefully watched; it may mean a relapse, which in my experience is not uncommon, or in a young horse it may indicate the advent of strangles.

Pulse.—I do not like to say that there is a characteristic pneumonic pulse, but there certainly is a pulse almost invariably associated with inflammatory chest trouble; it has been described as the oppressed pulse, and the name fitly describes its character. It is a pulse difficult to take, quick, and gives the feeling of being partly empty; after the more acute phases of the attack have passed away the pulse recovers a little of its character. But apart from the quality of the pulse, the number of pulsations is an excellent guide in gauging the progress of a case. They are usually between 60 to 70 beats per minute to start with, but I have met with them as high as 100.

In cases doing well a reduction in the heart beats will occur, and this reduction agrees in point of time with the fall of temperature. A drop from 60 to 55 is quite satisfactory; a rise of two or three pulsations at night during this period need create no alarm, for by the next morning the pulse has again dropped, and in this way from day to day, extending probably over a week, the pulse rate gradually falls until it reaches the normal.

I would, however, issue a word of caution with regard to moderately slow pulses during the period of invasion. Pulses of 45 and 48 are not uncommon at this time, but they must not cause us to relax our vigilance; in the same way they are not uncommon when the grey degeneration in the lungs is breaking down into abscesses. In either case, without previous warning, these low pulse rates are calculated to throw one off their guard.

Until my experience became extended, I should never have believed it possible for a horse to have pneumonia in the acute stage with a pulse of only 45 to 50. I now know it to be one of the most undoubted facts I have to record.

Respirations.—The breathing in pneumonia is quick, in some cases very quick, with nasal respiration; but it is a mistake to suppose that a hurried breathing is absolutely essential to the existence of pneumonia. I had two fatal cases where the respirations were 12 and 14 respectively, and such is really calculated to lead one astray. From 20 to 30 or 35 is the most common number of respirations per minute, though in a few cases they may be much higher. It will generally be found that if the respirations are rapid there is some degree of pleurisy present; it is the pain in pleurisy which hurries the breathing.

To a young practitioner I would say keep an eye on the respirations; your case is not out of danger despite the fall in pulse rate and temperature, until the breathing drops to nearly normal. To my mind, apart from auscultation, the most certain indication of a chest doing badly during the second and third weeks of the disease is rapid respiration.

The respiratory movements of pneumonia are full and true, that is to say, inspiration is deep, expiration well marked, and completed in a single movement. Occasionally towards the termination of a fatal case a double expiratory effort may be seen, but that is probably due to the weight of the hepatised lungs on the diaphragm, or to pneumothorax.

Nasal respiration need not be present; there need not be the least extra movement in the nasal opening, even with respirations of 30 per minute; on the other hand, towards the termination of a fatal case nasal respiration may be very evident. The amount of nasal movement indicates the amount of distress in breathing, therefore I am inclined to associate it with pleuritic complications rather than with pure pneumonia, but when existing in the latter disease it is always, I think, an unfavourable symptom.

Nasal Discharge.—A nasal discharge during pneumonia is common where bronchitis exists as a complication, and it is then of a muco-purulent character. In the early stages of acute pneumonia there may be a scanty amber-coloured discharge of serum, and, as previously noted, there is during resolution a free muco-purulent discharge during the removal of the lung exudate.

The Urine.—The urine in pneumonia may be acid or alkaline in reaction, depending upon the amount of fever, and whether the patient is feeding; if the appetite remains fairly good, and in many cases—I refer solely to young horses—it is all that could be desired, the normal alkaline reaction is obtained, as the patient is not living on its own tissues. The secretion frequently contains an undue amount of mucin; in one urine I examined it was like a jelly and poured out in clots. The specific gravity does not undergo any change, it being within the wide variations found in health. The colour is generally some tint of yellow, high-coloured or reddish urines being in my experience exceptional. Unless acid, the turbidity normal to urine remains.

There is, however, an abnormal constituent which is rarely absent, and that is albumen. Proteids, such as serum-albumen, globulin, and proteoses are almost invariably present, or, if the two former be absent, that intermediate product of albumen and peptone, viz., proteose, will almost invariably be present.

I have drawn attention to this previously, and pointed out its diagnostic value in incipient or doubtful pneumonia. I have treated horses for pneumonia on the strength of proteose in the urine, and have anticipated the attack, which, in my at present limited experience, has always invariably followed. I say limited, for the reason that the urine in all the cases which have come under my care has not been examined, but a sufficient number of observations have been made to warrant me in drawing particular attention to this pneumonic reaction in the urine.[1]

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[1] The test for proteose and proteid is nitric acid. To a little of the urine in a test tube add carefully a drop of nitric acid, and let it run down the side, so that the eye may catch the reaction on the acid meeting the urine. If *proteid* is present an opacity varying from a white cloud to a white precipitate is formed, and on boiling this is rendered still more evident. If *proteose* be present a delicate cloud or a well-marked opacity will result, and if this be boiled it entirely disappears, *to re-form on cooling*. When both proteid and proteose are excreted the latter is judged by observing the supernatant liquid before and during boiling, and after cooling. In boiling the urine of the horse with even one drop of HNO_3 the contents of the tube are often without warning ejected with considerable violence on arriving at boiling point. The observer should always turn the mouth of the tube away from himself.

If subsequent experience shows proteose to be present in other inflammatory diseases in the horse, its diagnostic value in pneumonia will be destroyed, while the fact of its presence remains; future investigation will settle the point.

I do not understand why proteid in any form should find its way through the kidneys in pneumonia. The kidneys give no naked-eye appearance of disease, yet albuminuria without a damaged epithelium in the uriniferous tubes is probably unknown. The point is one for the pathologist to settle.

With the advent of convalescence the proteid disappears from the urine.

The pneumonic urine does not contain sugar, but it contains a substance or substances which exert a considerable reducing effect upon salts of copper. I can afford no information on this subject, as I have only recently observed it.

It it said in the human subject that the chlorides disappear from the urine during the fever period, and return with convalescence. In the horse the chlorides may be reduced in amount, but they do not disappear. To test whether there is any reduction in chlorides the whole twenty-four hours urine would have to be collected, which is not under ordinary circumstances possible.

Relapses.—Relapses are by no means uncommon in pneumonia. They generally occur two or three days after convalescence appears to have been established, though I have had them happen much later than this. Such cases do not necessarily run again through the entire course of the disease, nor are they as a rule fatal.

VARIETIES OF PNEUMONIA.

Hitherto we have considered pneumonia as a disease in which the only variation is the intensity of the symptoms, but pathologists have quite another tale to tell us, and the varieties of pneumonia must briefly engage our attention.

I can bring no expert opinion to bear on this subject, not even the experience to be derived from a microscopical investigation of the diseased tissues; but I am justified in the remarks to be made, by knowing that they represent the views of an authority on pathological questions, Professor J. M'Fadyean—to whom I am indebted for much information, and the correction of erroneous ideas.

Pathologists describe three forms of pneumonia, viz., croupous, catarrhal, and interstitial; well-marked microscopical and macroscopical appearances distinguish these various forms, but so far as I know there is nothing specially diagnostic in their clinical features.

Croupous or Lobar Pneumonia.—In this there is a fibrinous exudation into the air cells of the lungs (hence the term croupous). The exudate often contains only a few corpuscular elements. During the progress of the inflammatory attack the lung passes through certain definite stages, viz., engorgement, red hepatisation, and grey hepatisation.

During engorgement the lung is filled with blood, and feels doughy, and on squeezing the cut surface blood and serum escape from it. The microscopical changes of this stage are engorgement of the capillaries of the air cell, with swelling and sometimes detachment of the alveolar epithelium.

In red hepatisation the lung is firm, closely resembling liver both in appearance and feel, and it sinks in water. The cut surface of the lung presents a finely granular appearance, due to the projection of minute fibrinous plugs from the air cell. Microscopically the alveoli are found filled with a solid exudate composed of fibrin, in which may be found a few corpuscular elements.

In grey hepatisation the lung tissue is greyish red or greyish yellow, firm but not so resisting to the feel as red hepatisation, and it still sinks in water. Microscopically it is found that the exudate no longer fills the cells, and is undergoing fatty or mucoid degeneration.

The description just given of the naked-eye appearances of croupous pneumonia, will strike the ordinary observer as corresponding very closely to, if not identical with, the lesions found in the horse. But, on the authority of M'Fadyean, croupous pneumonia in the horse is extremely rare, and what looks like red hepatisation in equine pneumonia is not what human pathologists regard as red hepatisation, though the naked-eye appearances suggest that no more fitting term than red hepatisation could be employed to indicate the appearance. In the same way, what might be taken for grey hepatisation is not what pathologists understand by that term.

All this is very puzzling, and for a long time I could not shake off the dogma of my early teaching, and even now it is convenient to speak of red hepatisation ; in fact I have done so throughout this communication, while instead of speaking of grey hepatisation I have described that as grey degeneration.

Catarrhal, Lobular, or Broncho-Pneumonia is a disease characterised by a proliferation and desquamation of the alveolar epithelium. The colour of the affected lung varies from greyish red to greyish yellow. The inflamed areas are in patches, generally sharply defined. The characteristic naked-eye appearance of this form of pneumonia is that when the portion of affected lung is incised and squeezed a thick yellowish or whitish material escapes from the smaller bronchi and air cells. Microscopically the alveoli are found to contain epithelial-looking cells derived by proliferation from the alveolar epithelium ; the exudation therefore is essentially cellular.

In the horse the common form of pneumonia is neither croupous nor catarrhal, but a special type which Professor M'Fadyean informs me is quite as distinct in its way as that of pleuro-pneumonia in cattle. As its chief characteristic is the tendency to necrosis the term necrotic pneumonia has been applied to it, and by some authorities this form of the disease is believed to be infectious, hence the term infectious pneumonia.[1]

It is this equine pneumonia with which I have been dealing throughout this communication. There may be more than one variety of it, at least, judging from the virulence of the attack one is sometimes inclined to think this may be the case, but greater virulence by no means necessarily indicates a new type or variety, and it may well be that it is the same disease which kills exceptionally in two or three days, and commonly in two or three weeks. From what we have previously said we would take exception to the term infectious pneumonia, while the only objection to the term necrotic pneumonia is that necrosis does not occur in every case, as judged by the fact that the recoveries from the disease are very much more numerous than the deaths.

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[1] See an excellent paper by Bartke in this Journal, Vol. IV., No. 4.

We may here conveniently and briefly consider the *post-mortem* appearances of the affection.

POST-MORTEM APPEARANCES.

The chief lesions in pneumonia are situated in the anterior and inferior lobes of the lung. I have seen the anterior lobes quite free, but this is most exceptional. The extent of the lesion depends as a rule on the length of time the case has lasted; large areas with long-lived cases, relatively small areas in short-lived ones, are the rule. If the lung be divided into three regions the same as the chest wall, we may say that it is common to find the whole of the inferior third affected, more rare to find two-thirds affected, and only once have I seen an entire lung attacked. Now, whereas in the lower lobes practically not a particle of healthy lung may be seen, in the middle regions islands of healthy lung are commonly met with, while even if the lung be affected from top to bottom there are still areas not touched by the disease. The inflammation runs up higher on the inner face of the lung than it does on the outer, so that on making a transverse section of the organ the affected area—which is sharply marked off from the unaffected,—runs obliquely across the lung from above downwards and outwards.

Even in those cases where the violence of the attack appears to be limited to the lower lobes of the lung, it is common to observe that the relatively healthy parts are mottled with patches of congestion, the areas being as large as a half-crown or a five-shilling piece.

The pneumonic lung is swollen, firm to the touch, and through the pleura can be seen to be intensely discoloured—red, crimson, or purple. On section it presents the character of a piece of liver, and is of much the same colour. On pressure but little fluid can as a rule be expressed from the cut surface; the tissue is very friable, and sinks in water. Even within the area of the greatest disturbance small islands of healthy lung may be seen.

The condition of lung cannot always be judged from the view it affords through the pleura; in the inferior lobes, it is true, it would hardly be possible to have the lung tissue affected without external evidence of the same; here the effect of gravity may explain the greater intensity of the vascular disturbance. But the higher one ascends in the lung the less marked is this, and considerable disease may be situated close to the surface of the lung, of which the view through the pleura does not give the slightest indication. I have once observed beneath the pleura a thin layer of healthy lung covering almost the entire organ, while beneath this again was the inflamed tissue.

If the process of hepatisation has gone on to a further stage, grey degeneration is met with, and the tissue is then greyish-white or yellow in colour, in either large or small areas, generally the latter. Each of the areas has an irregular ring around it, giving somewhat the appearance of a magnified nutmeg; in course of time the tissue breaks down and an abscess is formed.

The abscesses are situated most commonly in the anterior lobes, which may literally be so riddled with them as to defy the detection of any lung tissue; close to the bifurcation of the bronchi is another favourite place, and after this they may be found anywhere and everywhere, close together or far apart, one or two or dozens.

The contents of the abscesses are greyish brown, cheesy, grumous material, which under other circumstances would be called inspissated pus; occasionally I have found true laudable pus, but this is very uncommon. This cheesy material can be shelled out of its sac, and an irregular ragged cavity results lined by a membrane of a purplish hue, and in the case of large abscesses traversed like so many cords by the blood-vessels of the part, which are greatly thickened in their coats. Once only have I known them rupture and produce death by hæmorrhage. The largest abscess I have met with contained about half-a-gallon of cheesy degenerated tissue, and occupied the greater part of the interior of a lung. In such extensive abscesses everything disappears—vessels, bronchial tubes, etc., for on washing with a stream of water a complete cavity is left. The ordinary size of abscess is that of a walnut, and when the lung is riddled with them, and the section washed, the appearance is very characteristic. I once found the abscess cavities with contents resembling necrosed human skin.

In the upper part of the lung abscesses may be found in what practically amounts to healthy lung tissue. I believe these to be secondary abscesses, the result of infection.

The blood-vessels of the lungs as a rule contain firm fibrinous plugs, and for years I have considered that the plugging of the vessels may help to account for the necrosis.

It is not uncommon to meet with a little œdema beneath the pulmonary pleura, and in the anterior lobes, where marked interlobular tissue exists, œdema between the lobules is invariable.

The bronchial tubes may or may not be affected. The most intense pneumonia may exist without altering the colour of the bronchial mucous membrane; as a rule, however, the tubes are affected, being dark red in colour, with characteristic longitudinal stripings. When abscesses exist in the lung the tubes contain some of their contents.

The heart may be distended and contain fibrinous clots in the auricles and fluid blood in the ventricles, or firm clots in all four cavities, especially the ventricles; but I have found the heart contracted and the cavities empty, though this is certainly the exception. Sometimes the pericardial sac contains a little fluid.

In the condition I have endeavoured to describe, the pleura is perfectly healthy; if, however, an abscess approaches the surface of the lung, the pleura at the part is injected, thickened, and opaque, but the general pleural surface of both ribs and lung is absolutely healthy.

If before the lungs are removed from the body an attempt be made to distend them with bellows fixed in the trachea, it will often be found impossible to inflate them, a leak into the pleural cavity having occurred.

(*To be continued.*)

PARASITIC GASTRO-ENTERITIS IN SHEEP AND LAMBS.

By J. M'FADYEAN, Royal Veterinary College, London.

DURING the autumn of 1895 an unusually large number of lambs died on many farms in Lincolnshire, Leicestershire, Yorkshire, and other parts of England, from a disease which the previous experience of flockmasters did not enable them to recognise. The disease began towards the latter part of September, and it broke out almost simultaneously at places widely remote. It continued to carry off large numbers of lambs for several weeks, and declined about the end of October.

In anticipation of a recurrence of similar losses in 1896, a circular was addressed in October last to members of the veterinary profession practising in Lincolnshire and Leicestershire, requesting them to co-operate in an investigation by sending me the carcases or viscera of lambs dying in flocks under their care. This request was soon followed by the arrival of the specimens asked for, and during the following four weeks the viscera of about forty lambs were received at the Royal Veterinary College.[1] The main purpose of this article is to describe the result of the examination of this material, but before that is done it may be as well to describe briefly the symptoms exhibited by the diseased lambs.

Symptoms.—A knowledge of these has been gained from information supplied by those who forwarded diseased viscera, and from personal inspection of the live subjects on several farms.

In the first place, it is important to note that in all the cases that were brought under my notice in the autumn of last year the disease was confined to the lambs (about seven or eight months old), the ewes and other sheep over one year old escaping.

The symptom most frequently exhibited was diarrhœa, and this was associated with rapid loss of condition. The appetite was impaired, and towards the end almost entirely lost. Thirst, on the other hand, was increased, and in some cases a morbid propensity to lick sand or earth was observed. This, it may here be remarked, would appear to have been very commonly present, judging by the frequency with which sand and earth were found in the stomachs, at the *post-mortem* examination. The affected lambs had a dull dejected appearance, and the temperature was very constantly above the normal, varying from 105° to 108° F. There was no evidence of acute pain, and cough, as a rule, was not a symptom that had attracted attention.

The duration of the illness in fatal cases varied considerably. As a rule, the lambs were ill for several days or weeks, with the symptoms above described, before death took place, but sometimes the course of the disease did not exceed a day or two. The rate of mortality varied from 10 to 20 per cent. of the flock, and in many cases almost all the members of the flock were affected with the disease, or at least exhibited its most constant symptoms—diarrhœa and loss of condition.

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[1] I take this opportunity to thank the following members of the veterinary profession for the trouble they took in forwarding specimens :—Messrs Hoole, Sleaford; Kidd, Melton Mowbray; Holmes, Lincoln; Hackett, Spalding; Porter-Smith, Boston; Hicks, Metheringham; Clarke, Wragby; Thomson, Horncastle; Eve, Caistor; Field, Grimsby; Perry, Market Bosworth; Stevenson, Brigg; Lennox, Crowland; Leach, Boston; Freer, Uppingham; Clarke, Kirton-Lindsey; Gresswell, Louth; and Brooks, Leadenham.

Lesions.—The nature of the lesions which carried off so many lambs during last autumn—and presumably also in the autumn of 1895—will be gathered from the following abbreviated *post-mortem* records of thirty-nine cases.

No. 1.—There is a small area of consolidation at the apex of the right lung, and an adhesion between that and the pericardium. A considerable number of *Strongylus filaria* are present in the bronchi. The lung tissue is œdematous throughout. The fourth stomach contains a quantity of whey-like material, and microscopic examination of this reveals the presence of large numbers of small nematode worms.[1]

The mucous membrane of the fourth stomach and also that of the duodenum is somewhat congested. The mucous membrane of the large intestine is normal, but the cæcum contains considerable numbers of the *Trichocephalus affinis.* Other organs normal.

No. 2.—In each lung the apex and part of the lower edge opposite the heart are hepatised. The bronchi contain a large number of the *Strongylus filaria.* The mucous membrane of the fourth stomach is anæmic, and scrapings from it show numbers of small nematode worms. The duodenum is congested. The mucous membrane of the large intestine is normal, but a few *Trichocephali* are present in the cæcum.

No. 3.—The apex and the middle lobe of each lung are hepatised; there is also a small area of hepatisation in the base of the left lung. A few *Strongylus filaria* are present in the bronchi. The fourth stomach contains about half-a-pint of pultaceous earth (loam and sand). The mucous membrane of this stomach is anæmic; adherent to it there are a few worms belonging to the species *Strongylus contortus* and very numerous small nematodes. The duodenum is congested, and the cæcum contains a few *Trichocephali.*

No. 4.—Killed when unable to stand and evidently in a dying condition. The lungs are normal save for a small area of collapse in each, and only two or three *Strongylus filaria* are present in the bronchi. The mucous membrane of the fourth stomach is anæmic, and numerous small nematode worms are found on microscopic examination of scrapings from it. A considerable number of ova, some showing formed embryos in them, are also present. No parasites are found free in the stomach contents. At places throughout the small intestine the mucous membrane is deeply congested. The lining membrane of the large intestine is normal. The cæcum contains numerous *Trichocephali.* A few small nematodes are found on microscopic examination of scrapings from the small bowel. Other organs normal.

No. 5.—Both lungs are extensively hepatised; the consolidation involves the apex of each lung and a considerable part of the principal lobes. In the left lung there is a partially calcified nodule as large as a pea. No parasites present in the bronchi. The fourth stomach is at some places congested, at others anæmic. Adherent to the mucous membrane of the fourth stomach there are four worms belonging to the *Strongylus contortus*, and on microscopic examination of scrapings from the membrane numerous small nematodes are found. The duodenum is normal. At some places in the large intestine there is marked congestion of the mucous membrane; no *Trichocephali* present.

No. 6.—A number of areas of collapse are present in each lung, and the bronchi contain a considerable number of the *Strongylus filaria.* The fourth stomach contains about 2 ounces of semi-solid food, and its mucous membrane is slightly congested. Adhering to the mucous membrane there are four examples of the *Strongylus contortus*, and on microscopic examination very numerous small nematodes are found. The duodenum is more distinctly congested than the stomach. The large intestine is normal, and contains no parasites.

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[1] In this and the following cases, unless otherwise stated, "small nematode worms" means belonging to the new species hereafter described.

No. 7.—The right lung is extensively adherent to the chest wall, and about one-half of its substance is hepatised; the hepatised part on section is at some places of a uniform greyish-red colour, and at others it shows a reddish background finely mottled with grey. The bronchi of this lung contain a few examples of the *Strongylus filaria*. The mucous membrane of the fourth stomach is inflamed; it contains a few examples of the *Strongylus contortus* and very numerous small nematodes. The mucous membrane of the large intestine is much inflamed and shows a number of small hæmorrhages; a few examples of the *Trichocephalus affinis* are present, and a few small nematodes are found on microscopic examination of a scraping from the mucous membrane.

No. 8.—The lungs are normal save for some patches of collapse, and the presence of about two dozen examples of the *Strongylus filaria* in the bronchi. The mucous membrane of the stomach is inflamed, and it contains numbers of the small nematode worm. The duodenum is also inflamed, but to a less extent than the stomach. The large intestine contains a few *Trichocephali*.

No 9.—Both lungs are extensively hepatised, the consolidation involving the apex and the middle lobe of each lung; the hepatised parts are liver-coloured, and the pleura over them is covered with a small amount of fibrinous exudate. The bronchi in these parts contain thick muco-pus; in the large bronchi there are present about two dozen *Strongylus filaria*. The fourth stomach is anæmic; it contains small nematode worms. The small intestine is normal, as is also the large save for the presence of a few *Trichocephali* in the cæcum.

No 10.—The lungs contain several areas of hepatisation similar to those present in the preceding case, and in the same position. In each lung there is also a solid area in the basal lobe. No *filaria* are present in the bronchi. The stomach is slightly inflamed, and contains small nematode worms. The duodenum is normal. No parasites are present in the large intestine, which is also normal.

No 11.—Both lungs are normal, save for some small areas of collapse. No parasites are present in the bronchial tubes. The mucous membrane of the fourth stomach is anæmic, and microscopic examination of scrapings from it reveals the presence of small nematode worms. The duodenum is slightly inflamed. The mucous membrane of the cæcum is also slightly inflamed, and adherent to it there are a number of *Trichocephali*.

No 12.—The parts received are in an advanced state of decomposition. The lungs appear to be normal. The bronchi contain a few *Strongylus filaria*. The stomach has already been opened; its mucous membrane is anæmic, and covered with a material like thick black mud. On the mucous membrane there are present a few examples of the *Strongylus contortus*, and large numbers of small nematode worms.

No 13.—The apical lobe of the right lung, the lobe behind the heart, and about one-fourth of the main lobe are hepatised, and show slight pleurisy over the solid areas. On section the hepatised parts show a dark flesh-colour closely beset with greyish pinhead points. A muco-purulent fluid escapes from the bronchi of the solid areas on pressure; the large bronchi contain an abundant, frothy muco-purulent material, in which large numbers of the *Strongylus filaria* are present. The fourth stomach is empty; the mucous membrane is slightly inflamed, and large numbers of small nematode worms are found in scrapings from it. The duodenum is distinctly inflamed. Large intestine not sent.

No 14.—The lungs show some small areas of collapse along their lower edges, but no parasites are present in the bronchi. The fourth stomach shows bright vascular injection of its mucous membrane, this injection being unequally distributed; a scraping from it shows large numbers of the small nematode worms. The mucous membrane of the duodenum is diffusely inflamed. In parts the mucous membrane of the large intestine is also severely inflamed, and numerous *Trichocephali* are present.

No 15.—In each lung there are several hepatised areas. The large bronchi contain a considerable number of the *Strongylus filaria*, and their mucous membrane is congested. The fourth stomach, which is empty, shows slight congestion of its mucous membrane at some places; small nematode worms are found in scrapings from it. The duodenum is more deeply congested, especially in its first few inches. No parasites in the large intestine.

No 16.—The lungs are normal, save for a few *Strongylus filaria* in the bronchi and some congestion of the bronchial mucous membrane. The fourth stomach has already been opened; its mucous membrane is slightly congested, and scrapings from it show small round nematode worms. The membrane is largely coated with fine sand, and there is also a good deal of sand on the mucous membrane of the large intestine, which shows some congestion; a few *Trichocephali* are present.

No 17.—There are some areas of hepatisation in each lung, and the large bronchi contain abundant frothy mucus and a large number of the *Strongylus filaria*. The fourth stomach contains a small quantity of muddy liquid, and a good deal of sand adherent to the mucous membrane; the latter is anæmic, and scrapings from it show small round worms. The duodenum is also anæmic and a few *Trichocephali* are present in the cæcum.

No. 18.—The lungs are normal, save for some small patches of collapse; a few *filaria* are present in the bronchi. The fourth stomach contains about three quarters of a pint of thick muddy liquid, and a ball of wool as large as a pigeon's egg; the mucous membrane is anæmic, and scrapings from it show small round worms. No parasites present in the large intestine.

No. 19.—Lungs normal, and bronchi free from parasites. Stomach and duodenum show inflammation of the mucous membrane; small nematode worms are found on examination of a scraping from the former. A few *Trichocephali* are present in the large intestine.

No. 20.—Lungs normal. A few *filaria* present in the bronchial tubes. The fourth stomach is inflamed, but somewhat unequally; it contains small round worms. The duodenum is also inflamed. Other intestines not sent.

No. 21.—The lungs show a large number of the areas characteristic of the disease caused by the *Strongylus rufescens*. Numerous *strongylus filaria* are also present in the bronchi. The fourth stomach shows small round worms. Intestines not sent.

No. 22.—The parts received for examination are very putrid. Two small areas of consolidation are present in each lung, and a few *filaria* are present in the bronchi. The fourth stomach is anæmic, and scrapings from it show small round worms. No parasites in the large intestine.

No. 23.—The lungs are normal, and no parasites are present in the bronchi. The mucous membrane of the fourth stomach is deeply inflamed, and small round worms are found in scrapings from it. The mucous membrane of the duodenum is also slightly inflamed. The mucous membrane of the cæcum is severely inflamed, and numerous *Trichocephali* are adherent to it.

No. 24.—The apical lobe of the right lung is hepatised, and in both lungs there are small areas of collapse along the lower edge. The bronchi contain a large number of the *Strongylus filaria*. The lining membrane of the fourth stomach is inflamed, and numerous small round worms are found in scrapings from it. The mucous membrane of the large intestine is anæmic; very numerous *Trichocephali* present in the cæcum.

No. 25. The entire carcase of a lamb. The lung tissue is somewhat œdematous, otherwise it is normal, and no parasites are present in the bronchi. The fourth stomach contains about half-a-pint of muddy water, and its mucous membrane is anæmic. Small nematode worms are found in the gastric contents. The mucous membrane of the large intestine is pale; no parasites are present on it. Other organs normal.

No. 26. At the base of each lung there is a solid area, which on section shows the characters of catarrhal pneumonia. The bronchi leading into these hepatised parts contain a few *Strongylus filaria.* The fourth stomach is empty, and its mucous membrane is anæmic; scrapings from the mucous membrane show small nematode worms. Two examples of the *Strongylus contortus* are also present in the stomach, and a considerable number of *Trichocephali* are present in the large intestine.

No. 27.—In each lung there are some areas of hepatisation, and the air tubes are the seat of a purulent bronchitis, and contain numerous *Strongylus filaria.* The fourth stomach and the duodenum are inflamed, and scrapings from the former show numerous small round worms. The lining membrane of the cæcum is congested, and on it there is one small oval ulcer; a few *Trichocephali* are present in it.

No. 28.—Numerous *Strongylus filaria* in the bronchial tubes; lung tissue normal. Fourth stomach contains small nematode worms; the mucous membrane is anæmic. The duodenum is inflamed. Large intestine normal.

No. 29.—An area of hepatisation is present in one lung, and a considerable number of *Strongylus filaria* are present in the bronchi. Only a piece of the fourth stomach is sent, and scrapings made from this show small round worms. A few *Trichocephali* present in the cæcum.

No. 30.—The apex of the right lung is solidified, and a few *filaria* are present in the bronchi. The fourth stomach contains about half a-pint of thin muddy liquid, and some fine sand; also a packet of wool about the size of a pigeon's egg. The mucous membrane of the stomach is at some places inflamed; it contains a few specimens of the *Strongylus contortus* and numerous small round worms. A few *Trichocephali* are present in the cæcum.

No. 31.—Lungs normal; no parasites in the bronchi. Fourth stomach contains about half-a-pint of very muddy liquid, a few of the *Strongylus contortus* are present, and very many small round worms.

No. 32.—Lung tissue normal. Considerable number of *Strongylus filaria* in the bronchi. About half-a-pint of muddy liquid in the fourth stomach; small round worms very numerously present in it.

No. 33.—Lung tissue normal. Numerous *Strongylus filaria* in the bronchi. Stomach contains a quantity of muddy liquid, and its mucous membrane is anæmic; small round worms are present in it. Mucous membrane of the duodenum also anæmic. No parasites in the large intestine.

No. 34.—Lungs normal, save for the presence of a few *Strongylus filaria* in the bronchi. The mucous membrane of the stomach is normal. After a long search of both contents and mucous membrane only two parasites were found, one being a male *Strongylus contortus* and the other a small female round worm. Small hæmorrhages are present on the mucous membrane of the large intestine, but no parasites are found here.

No. 35.—Lungs normal; no parasites in the bronchi. A scraping from the mucous membrane of the fourth stomach shows small round worms. No parasites in the large intestine.

No. 36.—In each lung there is a considerable area of hepatisation opposite the heart, and a few *Strongylus filaria* are present in the bronchi. Close to the pylorus the mucous membrane of the fourth stomach is inflamed, but elsewhere it is anæmic; it contains numerous small round worms. The lining membrane of the duodenum is much inflamed. The mucous membrane of the cæcum is also intensely inflamed, and shows minute hæmorrhages; no *Trichocephali* are present in it.

No. 37.—The entire carcase of a lamb; very putrid before delivery. Lungs normal, save for the presence of a few *Strongylus filaria* in the bronchi. Fourth stomach appears to have been inflamed; a scraping taken from its lining membrane shows a large number of small round worms. The duodenum is also distinctly inflamed. No parasites in the large intestine.

No. 38.—Lungs normal. Bronchi contain a few *Strongylus filaria*. Fourth stomach nearly empty; its mucous membrane is at some parts distinctly inflamed, and microscopic examination of scrapings from it shows large numbers of small round worms. The mucous membrane of the duodenum is inflamed throughout. Mucous membrane of the large intestine is also inflamed and it shows some small hæmorrhages into its substance. No parasites in large intestine.

No. 39.—Entire carcase of a lamb. The bronchi contain a considerable number of the *Strongylus filaria*. There is a small hepatised area at the apex of the right lung. Fourth stomach contains about 4 ounces of turbid liquid, in which very numerous small round worms are present. The mucous membrane of the fourth stomach and that of the duodenum is intensely inflamed. The cæcum is almost normal, and no parasites are present in it.

Cause of the Disease.—The most constant abnormality in the foregoing series of cases was the presence of small nematode worms in the fourth stomach, and at an early stage in the investigation the conclusion that these parasites were the main cause of the mortality was arrived at. It will be observed, however, that in a considerable number of cases, other parasites were simultaneously present, and it becomes necessary to estimate how far these were responsible for the death or illness of the lambs in which they were found.

The *Strongylus filaria* was present in the bronchi in thirty of the cases, and in eighteen of these the lungs were the seat of lesions that were presumably caused by that parasite, and which must have contributed to the animal's illness. In only a few cases, however, was the condition of the lungs such as to afford a reasonable explanation of the lamb's death, and in more than half the cases the lungs were either healthy, or showed only trifling lesions. It, therefore, cannot be held that the outbreaks of last autumn were ascribable to the common "lung worm."

The *Strongylus rufescens* was present in only one case, and it is therefore unnecessary to consider it further.

The *Trichocephalus affinis* was present in eighteen of the cases, but save in six of these it was not numerous. In these six cases it may have been partly responsible for the diarrhœa, but it cannot be credited with having played an important part in the outbreaks.

The *Strongylus contortus* was present in only eight instances, and even in these cases the number of worms present was too small to justify the conclusion that they had in any material degree been responsible for the illness of the animals in which they were found.

The small nematode worms appeared to be the one constant factor in the disease and in most cases the organ in which they were found was manifestly abnormal. The abnormality generally took the form of gastritis with inflammatory congestion of the mucous membrane, but in a considerable number of cases the lining membrane of the stomach was markedly anæmic. The parasites were generally in such numbers as to make it reasonable to ascribe the abnormalities of the lining membrane of the stomach to their presence, while the condition of the stomach was sufficient to account for the symptoms of the disease.

In a large number of cases the irritation of the mucous membrane was continued into the duodenum, but, as a rule, the remainder of the small intestine was not inflamed. Occasionally the mucous membrane of the cæcum or colon was inflamed, and this even when only a few *Trichocephali* were found there.

Although the worms are not strictly speaking of microscopic dimensions, it is impossible to detect them with the naked eye when they are suspended in the stomach contents, or lying on the gastric mucous membrane. They are readily detected when a scraping from the surface of the mucous membrane is spread on a slip of glass and viewed under a low magnification. Numerous eggs, and occasionally also free embryos, are found along with the worms in the gastric contents. When the stomach is opened immediately after death, the living active worms are most numerous on the mucous membrane, but when decomposition sets in the parasites soon die and begin to shrivel up. They may then be found in great numbers in the stomach contents.

Characters of the Parasite.—To the naked eye the colour of the worms is a dull white. The males are smaller and less numerous than the females.

The females found in the stomachs of lambs in October and November were about 10 mm. long. Those shown in Fig. 2, Plate I., may be taken to represent the adult size of the parasite. They were taken from a lamb that was brought in November last from a farm on which the disease was prevalent, and kept at the Royal Veterinary College till the 29th of January, when it was killed. In this lamb the worms were very uniform in size, the females being about 12 mm. long, while the length of the males was about 8 mm. The largest female worms are about ·13 mm. broad in front of the vulva. The breadth of the largest males in front of the bursa is about ·11 mm. In both sexes the integument carries fine transverse rings, and it also shows longitudinal ridges, of which from 10-12 are visible on either aspect of the body (upper or lower). About ·35 mm. behind the anterior extremity the skin carries on opposite sides of the body two small barb-like papillæ with a backward direction.

The female worms are thickest in the neighbourhood of the vulva, and taper towards either extremity; the tail or posterior extremity is sharp, and straight or slightly curved. The mouth is unarmed, and conducts into an œsophagus which has a thick muscular wall posteriorly. The intestinal tube takes a nearly straight course through the body, and the anus opens about ·14 in front of the tip of the tail. The vulva opens about one-sixth of the total length of the body from the posterior extremity. There are two uteri opening into opposite ends of a short vagina. The ovary of the anterior uterus commences a little behind the posterior end of the œsophagus, and its oviduct and uterus are continued directly backwards to enter the anterior extremity of the vagina. The second ovary begins a short distance behind the first, and its oviduct is continued past the vulva to join its uterus in the posterior part of the body. A short distance in front of the anus this uterus is sharply reflected forwards to open into the posterior extremity of the vagina. The full-sized eggs measure about 0·09 in length by 0·05 in breadth at their widest part. Those nearest the vagina are generally in the morula stage of division, but sometimes they show the outline of the embryo, and in a few cases fully-formed active embryos have been seen in eggs within the parent worm. The vulva is guarded by a flap-like cutaneous appendage, but this varies greatly in its degree of development, and in many worms it is indistinct or absent.

The bursa of the male worm is difficult to spread. When fully expanded it has the outline shown in the accompanying figure. The surface of the bursal membrane in the full-sized worms shows an irregularly tessellated pattern. Its greatest transverse diameter in the largest individuals is about 0·46 mm. The posterior rays spring from a common stem, and each is divided at its tip into two unequal branches. A little below it gives off on its outer side a short blunt branch. This mode of branching of the posterior rays recalls the appearance of a stag's horn, and it has suggested the name *Strongylus cervicornis*, by which I propose to designate the species. On each side the bursa is supported by six other rays, the characters of which will be much better gathered from the accompanying figure than from any verbal description. The two yellow spicules are a little over 0·2 mm. long in the largest worms; they taper slightly towards their free extremities, which have the appearance of being fissured longitudinally.

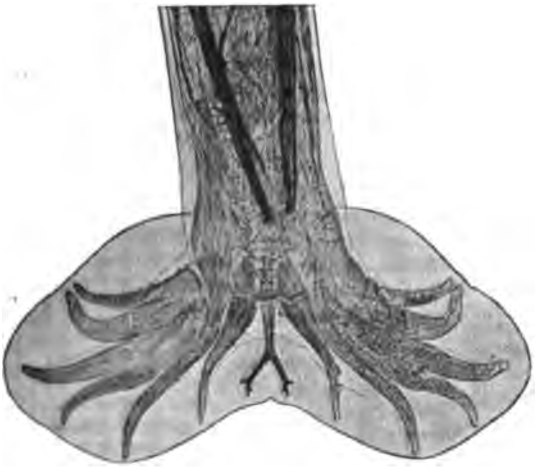

Fig. 1.—Caudal extremity of male *Strongylus cervicornis* (magnified).

Since it was announced in November last that the cause of the then prevailing lamb disease was a nematode worm belonging to a hitherto undescribed species, the view has been forward that the worm in question does not constitute a new species, but represents a stage in the development of the *Strongylus contortus*. That view must, therefore, be here examined.

Needless to say, when these worms were first discovered in the series of *post-mortem* examinations previously described, the possibility of their belonging to the *Strongylus contortus* or to some other species of strongyle previously observed as a parasite of the alimentary canal of the sheep, was carefully considered. So far as is known to me only three species of strongyle have up to the present time been reckoned among the gastro-intestinal parasites of the sheep, viz., the *Strongylus contortus*, the *Strongylus ventricosus*, and the *Strongylus filicollis*.

The *Strongylus contortus* is the best known of these, and the only one of them that is credited with being pathogenic. As has previously been mentioned, this worm was sometimes found in association with the smaller nematodes in the lambs examined last autumn, but never in such numbers as to warrant one in crediting it with having been in any material degree responsible for the illness of the animals in which it was found. That it is sometimes the cause of serious losses among young sheep and lambs is a well accredited fact. Outbreaks due to this cause have frequently been noted in Germany (Magenwurmseuche), and Simonds, Williams, and others have ascribed similar effects to the parasite in England and Scotland.

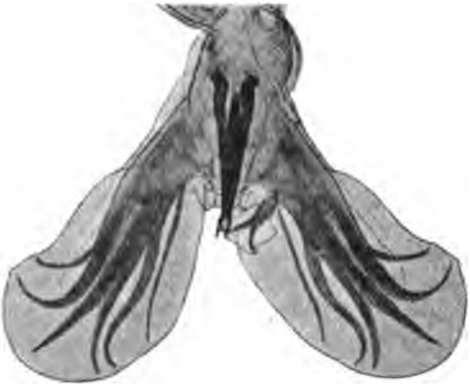

FIG. 2.—Caudal extremity of male *Strongylus contortus* (magnified).

The *Strongylus contortus*, as it is usually found, is a worm of considerable size, the females being from an inch to an inch and a quarter, and the males about half an inch in length. Two female worms are reproduced of exactly natural size in Fig. 1, Plate I. Throughout the greater part of its length the female has the appearance of a thread compounded of a white and a reddish strand, the former being wound spirally around the latter. The red central strand is the intestine, and the spiral white strand is composed of the two genital tubes. This very remarkable anatomical peculiarity makes it easy to distinguish the female *Strongylus contortus* at the very first glance, and the male worm has characters which are scarcely less distinctive. The accompanying figure (Fig. 2) reproduces a photograph of the magnified caudal bursa of a male worm of this species, and makes it possible to dispense with a detailed description of that part. It will be observed that the pouch has two distinct lobes, and that one of these lobes carries an asymetrical appendage, which represents a rudimentary third lobe, and is supported by the posterior ribs. The latter spring from a common stem, and each is divided for a very short distance at its tip.

Now, whether the comparison be instituted between the male or the female worms, it cannot be maintained that there exists between the *Strongylus contortus* and the parasite previously described under the name *Strongylus cervicornis*, a resemblance so close as to suggest that they belong to the same species. In fact quite the contrary is the case, for their characters are so dissimilar as to suggest that the worms belong to different species. The suggestion that they belong to the same species must mean that the worms here described as a new species were young and therefore imperfectly developed examples of the *Strongylus contortus*, which had not had sufficient time to assume the adult size and structure. Against this view there stands, in the first place, the fact that a few fully developed examples of the *Strongylus contortus* were in several instances found in the same stomach alongside of much more numerous small worms with the distinctive characters previously described. It might still be sought to explain this occurrence by supposing that there had been two distinct invasions separated by a considerable interval of time, and that only the worms of the first invasion had had time to grow to adult size. The following facts, however, render that view untenable. In November last a lamb from a flock in which many deaths from the small stomach worms had recently taken place was brought to the Royal Veterinary College, and it was kept there till the 29th of January, when it was killed in order to ascertain what changes had in the interval taken place in the size or structure of the parasites. The *post-mortem* examination showed that the worms were scarcely if at all larger than those that had been found in the stomachs of lambs of the same flock in November, though they were rather more uniform in size. In structure, however, they were quite unaltered. The worms shown in Fig. 2, Plate I., are from this lamb. It seems fair to assume that the worms found in this lamb on the 29th of January were in the animal's stomach when it came to the College ten weeks previously, and it only remains to consider whether that is a sufficient period to allow of complete development of the *Strongylus contortus*. That point is disposed of by Fig. 1, Plate I., for the two adult female worms there produced were taken from a lamb that was under ten weeks old. These worms must therefore have attained the adult size and characters in less than ten weeks, and probably in less than two months, for it is unlikely that infection took place during the first two or three weeks of life, at which time the mother's milk was the only sustenance of the lamb.

But even without this evidence it would have been justifiable to regard the small worms as a species distinct from the *Strongylus contortus*, for it appears in the highest degree improbable that the bursa reproduced in Fig. 2 could have had the structure shown in Fig. 1 for one of its developmental phases.

On similar grounds it is impossible to believe that the worms here described belong to the *Strongylus ventricosus* or the *Strongylus filicollis*. The first of these approaches to the *Strongylus cervicornis* in size, but it is distinguished from the latter by having its cephalic extremity expanded, and by having fourteen longitudinal cutaneous ridges at unequal distances apart, as well as by the form of its spicules and bursa.

The *Strongylus filicollis* is rather larger than the worm here described, and, among other characters that distinguish it, it possesses a cutaneous expansion at its anterior extremity, long slender spicules, and a bursa quite unlike that shown in Fig. 1.

As has previously been mentioned, in the outbreaks investigated in October and November last lambs alone were attacked, but since the beginning of the present year three very serious outbreaks of parasitic gastro-enteritis in ewes have come under my notice. In each of these the parasites belonged to the *Strongylus cervicornis*, but in two of the outbreaks they were accompanied in considerable numbers by the species which was described in the last number of this Journal,[1] as being sometimes responsible for outbreaks of gastro-enteritis in the ox, and for which the name *Strongylus gracilis* was suggested. In a fourth outbreak which has come under observation during the present year, in sheep a little under a year old, this last parasite was the only one present.

In two of these flocks the ewes had dropped lambs about Christmas, and in one of them there was a very heavy mortality among the lambs from the same cause during the latter part of February and the beginning of March. Both lambs and ewes suffered from diarrhœa and rapidly lost condition, and *post-mortem* examination of several animals showed that there was severe gastritis, with enormous numbers of small strongyles in the stomach.

The fact that sheep over a year old are seldom affected indicates that age brings a greater power of withstanding the injurious effects of the worms. The exceptional cases in which ewes were attacked may have been due either to an unusual degree of contamination of pasture or water, or to an enfeebled state of the body during lactation.

In each of these cases the disease was a new experience to the owner of the flock, although sheep had for years been kept on the farm.

Since the discovery of this species of worm last autumn it has in several instances been found in the fourth stomach of lambs that had died from other diseases than gastro-enteritis; in one case it was found in an experimental lamb which had been over three months at the College, and the flock from which this lamb came had not suffered from gastro-enteritis, nor had the lamb itself shown any symptom of that disease. In these cases, however, only a few worms were found, and their presence, unassociated with obvious symptoms or lesions, is in no way opposed to the view that the parasite was the main cause of the deaths among lambs last autumn. The *Strongylus filaria* is very frequently met with in the bronchi of apparently healthy lambs, but no one on that account denies pathogenic properties to the parasite. It is extremely probable that further observations will show that the new species is a very common inhabitant of the stomach of the sheep. Probably when it is present only in small numbers its effects are inappreciable, but when large numbers gain access to the stomach it becomes a formidable parasite.

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[1] Since the article referred to appeared my attention has been called to the fact that in the Agricultural Student's Gazette for August 1893, Mr Allen Harker described and figured a strongyle which had been found by Mr. W. T. Wilson in young cattle suffering from diarrhœa and wasting. This appears to me to be the worm for which I suggested the name *Strongylus gracilis*, but if that supposition is correct the posterior ribs of the bursa are inaccurately represented in Mr Harker's illustration.

Prevention.—The circumstances that favour the introduction of these parasites into the alimentary canal of the sheep are very obvious. The fæces of every animal that harbours the parasite contain eggs and embryos, and these are distributed on the herbage or soil, to undergo a degree of development at present unascertained. The young worms are likely to be taken in by sheep or lambs put to graze on such contaminated pasture, and the dirtier the land —that is to say, the greater the number of sheep kept on a given area—the greater will be the likelihood that a large proportion of the animals will take in what may be termed the pathogenic number of the parasites. There can be no doubt that overstocking of the land with sheep is one of the main factors in determining such outbreaks as prevailed last autumn.

Whether water is to any extent the vehicle of infection, or whether wet seasons are specially favourable for the development of the disease, is uncertain. It is true that the outbreak of last autumn was preceded by a period of heavy rainfall, but the summer and autumn of 1895 were comparatively dry, and yet it is not improbable that the heavy losses of that year were due to the same parasite. In one of the outbreaks previously referred to in ewes the water supply was bad, the sheep having to drink out of stagnant ponds, and in a second outbreak the ewes had been grazed on a common, part of which was wet and marshy. In the third case in which ewes were attacked the circumstances pointed very strongly to flooded land as the source of the disease. In this case only one out of three lots of ewes on the same farm was affected. All the ewes had been grazed together till the end of November, when they were divided. One lot, numbering fifty-eight, was put to graze on a meadow, which, during the time the ewes were on it, was partially flooded. About six weeks after the flood the disease manifested itself in this lot, and twenty-two of them died. Almost every ewe in the lot appeared to be affected, but no sign of the disease was manifested by the other ewes, which had been kept in common with the fifty-eight up till the end of November.

Although the cases just cited do not prove that water is the vehicle by which the worms are generally taken into the stomach, it would appear to be a wise precaution to prevent access to recently flooded land, or to stagnant ponds or ditches, and to provide well water where a clear running stream is not available.

From what is known of the life history of other nearly related species, it may be concluded that these parasites do not multiply during their period of sojourn in soil or water. Such free, sexually immature worms as do not succeed in finding entrance to the stomach of a sheep (or other animal) must perish without having added to the number of their species. How long such worms can maintain their existence outside the body is at present unknown, but it is improbable that the period extends to years. Hence, even badly contaminated land would probably become healthy again if sheep were kept off it for a year or two.

In the absence of direct observations on the point, one can only speculate regarding the efficacy of such agents as lime or salt when applied to the land with the object of killing the worms, but if the parasites are anything like so resistant in their earlier stages as they are when full grown it is difficult to believe that any chemical substance not absolutely destructive to the herbage could be relied upon to kill the worms in the soil.

Treatment.—Advantage was taken of the opportunity presented by the *post-mortem* examinations made last autumn, to test the killing power of some of the agents in most common use against worm disease in calves and lambs.

The general plan of the experiments was as follows. The worms were taken from the lungs or stomach of an animal immediately after death, and put into clean tap water. Their vitality was then tested by gently raising the temperature of the water to body heat, when the worms at once displayed very active movements.

The killing effect of the various substances mentioned below was tested by pouring a small quantity of it into a watch glass, and transferring to this a number of active worms from the warm tap water. At intervals some of the worms were re-transferred to warm water, and observed under the microscope in order to ascertain whether they were still alive.

Experiments with Nematode Worms (Strongylus convolutus?) from the Fourth Stomach of a Cow.

Placed a number of worms in ½ per cent. carbolic acid solution in water; jerky movements almost immediately after introduction. Removed some of the worms after five minutes into tap water slightly warmed; distinct movements on the part of some of the worms.

Put a number of worms into ½ per cent. solution of lysol in water; immediately after their introduction the worms exhibited slow bending movements in the fluid. These movements continued for five minutes; when they had ceased, the worms were transferred to warm water, in which the movements were resumed.

Put a number of the worms into 1 per cent. solution of carbolic acid in water; slow bending and unbending movements; after three minutes, movements slightly jerky. Transferred after five minutes to warm water, in which the movements were continued more actively.

Parallel experiment to the preceding with 1 per cent. solution of lysol in water. Movements stopped after two minutes; transferred to warm water at the end of five minutes; worms remained motionless.

Put a number of worms into Fowler's solution diluted with forty times its volume of water. After five minutes removed the worms to warm water, in which active movements were resumed.

Tested the effect of ½ per cent. carbolic acid, ½ per cent. lysol, 1 per cent. carbolic acid, and 1 per cent. lysol, after half-an-hour's exposure. The worms that had been exposed to ½ per cent. carbolic acid, and also those that had been in the ½ per cent. lysol, exhibited active movements, but those that had been in the 1 per cent. lysol and the 1 per cent. carbolic acid remained motionless.

Put a number of worms into turpentine well shaken with forty times its volume of milk. Transferred the worms to warm water after five minutes; very vigorous movements were exhibited.

Tested the effect of the diluted Fowler's solution (1 in 40) after half-an-hour's exposure; the worms exhibited active movements when transferred to warm water.

Put a number of worms into a saturated solution of chloroform in water. The worms soon become motionless, and when transferred at the end of five minutes to warm water they remained motionless.

Worms that had been for twenty minutes in the turpentine mixture (1 to 40 of milk) were found to be very active when transferred to warm water.

Left a number of worms overnight in a ½ per cent. solution of lysol, 1 per cent. solution of lysol, ½ per cent. solution of carbolic acid, 1 per cent. solution of carbolic acid, saturated solution of chloroform in water; Fowler's solution 1 to 40 of water, and turpentine 1 to 40 of milk. When tested at the end of twelve hours it was found that the worms in the ½ per cent. lysol, the 1 per cent. lysol, and the chloroform water were all dead. A few of the 1 per cent. carbolic acid worms were dead, but some were active. The worms from the other solutions were all active when transferred to warm water.

Experiments with the Strongylus filaria.

Put a number of worms into Fowler's solution diluted with nine times its volume of water, and examined them at intervals by transferring them to warm tap water. The last observation was made five hours and a half after the worms had been put into the solution, and at that time they were still quite active.

Put some worms into Fowler's solution diluted with four parts of water; when transferred to warm water the worms exhibited active movements after one hour and nine minutes (latest observation made).

Put a number of worms into undiluted Fowler's solution; all the worms exhibited active movements when transferred to warm water at ten minutes, and fifty-nine minutes afterwards (later observations not made).

Put a number of worms into 1 per cent. solution of lysol in water; in this they exhibited at first slow movements, but these ceased in three minutes. When transferred to warm water at the end of ten minutes they were found to be motionless, and continued observation showed that they were dead.

Put a number of worms into 1 per cent. carbolic acid in water; when transferred from this to warm tap water after ten minutes they all exhibited active movements. When examined one hour and eight minutes afterwards they were still active (latest observation made).

Put a number of worms into a saturated solution of chloroform in water; all movement on the part of the worms ceased in two minutes, but when transferred to warm tap water at the end of ten minutes the worms gradually became active again. They were found to be still alive and active three hours and sixteen minutes after removal from the chloroform water (last observation).

Put a number of worms into turpentine well shaken up with twenty times its volume of milk; the worms exhibited active movements when transferred to warm water at the end of ten minutes, and they were still active one hour and twenty-three minutes later (last observation).

Experiments with Small Nematode Worms (Strongylus cervicornis) from Lamb's Stomach.

Put a number of worms into 1 per cent. solution of carbolic acid in water; the worms all exhibited active movements when transferred to warm water at the end of ten minutes. When examined at intervals, up to two hours afterwards, they were still active.

Put a number of worms into 1 per cent. solution of lysol in water. All the worms were motionless when transferred to warm water at the end of ten minutes, and subsequent observation proved that they were dead.

Put a number of worms into undiluted Fowler's solution. All the worms exhibited active movements when transferred to warm water at the end of ten minutes, and these movements were still continued two hours and eighteen minutes later (last observation).

Put a number of worms into 4 per cent. solution of mercuric chloride in water raised to the body temperature; at the end of ten minutes many of the worms were moving a little in the mercuric solution; at the end of fourteen minutes some of them were still active; at the end of nineteen minutes they were all motionless, and when transferred to fresh warm water they were found to be dead.

Put a number of worms into a mixture of 1 of turpentine and 25 of milk, well shaken together. When tested at intervals, up to one hour and sixteen minutes afterwards (last observation), the worms were found to be still active.

It must be admitted that these experiments are calculated to give a shock to the confidence which has hitherto been very generally placed in some of the parasiticides mentioned. Turpentine is a sheet-anchor in the treatment of diseases caused by the round worms, and there appears to be a considerable body of clinical evidence in its favour, but it will be observed that in the strength of 2½ per cent. (with milk) it appeared to have no serious effect on worms (*Strongylus filaria*) that had been immersed in it for over twelve hours. Even with double that strength it had no apparent effect after a two hours' exposure. In practice it would be impossible to insure a two hours' contact between worms in the stomach of a living sheep and a 5 per cent. solution of turpentine, because of the necessity of considerably diluting the turpentine for administration, and the rapid absorption that sets in as soon as it comes in contact with the mucous membrane of the stomach. It need hardly be pointed out that if the circumstances are so unfavourable for the insuring of contact between a sufficiently concentrated solution of turpentine and stomach worms, the difficulty is immensely greater in the case of parasites lodged in the bronchial tubes or lung tissue.

Arsenic also appears to be a very weak parasiticide, since twelve hours' exposure to Fowler's solution diluted with forty times its bulk of water left the *Strongylus filaria* still active. This represents a strength of 1 grain in 10 ounces, and a stronger solution could not with safety be kept in contact with the mucous membrane of the stomach for anything like twelve hours. Besides, it will be noticed that even undiluted Fowler's solution (4 grains to the ounce of

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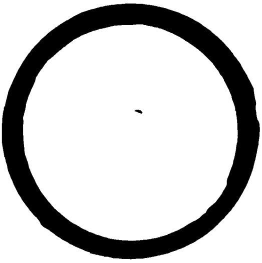

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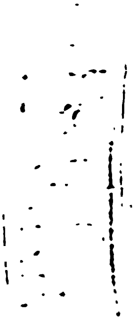

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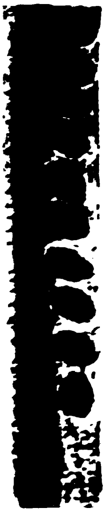

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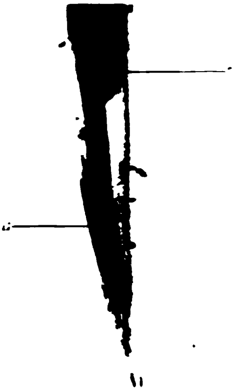

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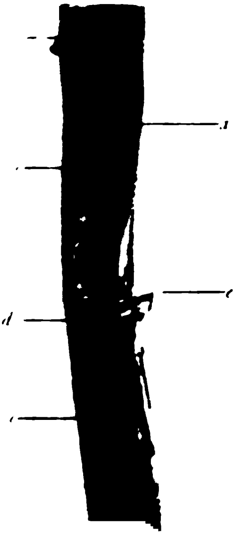

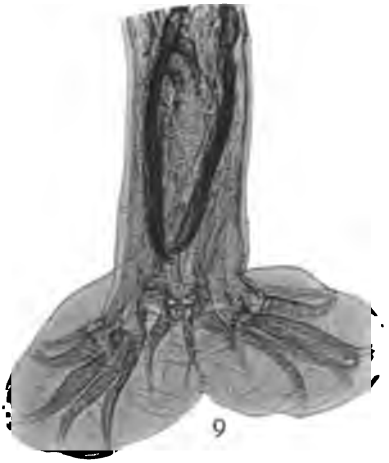

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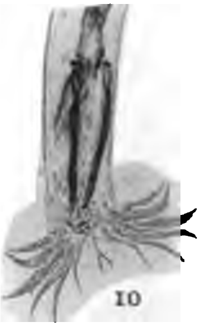

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liquid) did not kill stomach worms with a two hours' exposure, and 1 ounce of Fowler's solution is a poisonous dose for a lamb.

Carbolic acid and chloroform appear to be much more injurious to adult nematode worms than turpentine or arsenic, but less active than lysol. In each of the series of experiments a 1 per cent. solution of this substance in water proved fatal to the worms in ten minutes, and since half-a-pint of such a solution may with safety be given to a lamb of six months old, the experiments hold out a strong hope that lysol may prove valuable in the treatment of parasitic gastritis.

DESCRIPTION OF PLATE I.

Fig. 1. Two female worms of the *Strongylus contortus*, from the fourth stomach of a ten-weeks-old lamb. Natural size.

Fig. 2. Four worms of the *Strongylus cervicornis* from the fourth stomach of a lamb killed after it had been ten weeks at the Royal Veterinary College. The two smaller worms are males, the two larger females. Natural size.

Fig. 3. Integument of female *Strongylus cervicornis* (x 164).

Fig. 4. Body of female *Strongylus cervicornis* above the vulva (x 105).

Fig. 5. Caudal end of female *Strongylus cervicornis* (x 105); *a*, intestine; *b*, loop of posterior uterus.

Fig. 6. Body of female *Strongylus cervicornis* at vulva (x 105); *a*, egg in vagina; *b* and *c*, ends of uteri; *d*, integumental flap over vulva.

Fig. 7. Cephalic end of female *Strongylus cervicornis*, showing barbs (x 164).

Fig. 8. Body of female *Strongylus cervicornis* (x 105); *a*, intestine; *b*, eggs in anterior uterus; *c*, *c*, eggs in posterior uterus; *d*, vagina; *e*, vulva.

Fig. 9. Caudal end of male *Strongylus cervicornis* (x 164). The tips of the posterior rays have not been in focus.

Fig. 10. Same as Fig. 9, but from a different individual (x 130).

EDITORIAL ARTICLES.

PRACTISING PHYSICIANS AND ANIMAL TUBERCULOSIS.

So much has already been said and written regarding animal tuberculosis, and particularly with reference to its suppression, that it is difficult for any one to develop original views on the subject, but it cannot be denied that considerable novelty attaches to the method of dealing with this disease which has recently been propounded by a member of the medical profession. Sir James Sawyer, a Birmingham physician, has conceived the idea that "chambers of agriculture and allied bodies throughout the country should invite practising physicians, or other medical men practically interested in bacteriology, to meet assemblies of agriculturists, landowners, farmers, stockbreeders, farm labourers, and others, to explain, *viva voce*, the simple facts of the essential causation of tubercular diseases in man and in animals generally, with the view of taking many easy and common sense precautions for checking the spread and ravages of Koch's bacillus." The names of the classes to be convened for these courses of instruction make it clear that it was animal tuberculosis that was to be the main theme, for it cannot be held that the classes mentioned are in any special degree the victims of tuberculosis. The scheme appears to have had its inception in the belief that human phthisis must be attacked by endeavouring to check the spread of animal tuberculosis, and that practising physicians are the men most competent to give advice and directions with this object to those who have to do with cattle. It will be observed that practising physicians are grouped with "other medical men practically interested in bacteriology," in such a way as to suggest that the bulk of those who practise medicine at the present time take a practical interest in bacteriological work. Presumably this acquaintance with bacteriology on the part of the medical practitioner, coupled with the fact that animal tuberculosis is the great source of human phthisis, is thought to justify the intrusion of the "practising physician" into the field of the practising veterinary surgeon.

We hardly think that Sir James Sawyer's suggestion is likely to find much support from the Chambers of Agriculture and other allied bodies to which he appeals. Common sense is opposed to the idea that medical men are better fitted than veterinary surgeons to confer with stockowners regarding the best methods of dealing with tuberculosis among animals. Human and veterinary medicine are, no doubt, divisions of one great science, but knowledge of a part does not imply an acquaintance with the whole. The public will be best served, and good feeling between the two professions will be best maintained, by allowing the practising physician to attend to the ailments of his own species, and leaving to the veterinary surgeon the treatment of the diseases of the lower animals. If the experience of medical men with regard to disease of the human subject can throw any light on the prevention or treatment of disease of the lower animals, veterinary surgeons are ready to profit by it; but we doubt whether the average practising physician is in a position to instruct either the veterinary surgeon or the farmer as to the means of dealing with animal tuberculosis. To do the medical practitioner justice, we think that, as a rule, he would decline the task. Sir James Sawyer is the exception, for, following precept by example, he undertook to instruct the Warwickshire farmers as to what measures should be employed for dealing with tuberculosis "in the living animal and in the carcases of animals." We cannot say that the experiment has been encouraging. Sir James Sawyer is anxious to persuade stockowners that an animal "in perfect general and local health, as to structural integrity and vigour, is practically immune, under favourable conditions, against the deadly tubercle bacillus." Now, this statement contains just enough truth to make it more mischievous than an assertion wholly unfounded.

We know that an attack of any bacterial disease is a battle in which the issue is partly dependent on the powers of defence possessed by the animal attacked. Hence, an animal in vigorous health stands a better chance of resisting infection than one from any cause enfeebled. But that is a very different thing from saying that a healthy animal is immune against tuberculosis.

It would be interesting to learn on whose authority the assertion we have quoted was made. Who is the person who has made the observation that an animal in perfect general and local health is practically immune against the tubercle bacillus? For that matter, it may be asked where animals "in perfect general and local health, as to structural integrity and vigour," are to be found! We take it, however, that this was only Sir James Sawyer's way of saying that a healthy, vigorous animal is practically immune against tuberculosis, and, we assert, in contradiction of it, that an animal may exhibit every symptom of health and vigour, and yet be readily infected with tuberculosis.

The greatest difficulty that veterinary surgeons have to deal with in instructing stockowners regarding the means of preventing tuberculosis is to persuade them that the disease is infectious, and that no progress towards stamping out can be made as long as diseased animals are allowed to cohabit with the healthy. To tell them that healthy, vigorous animals are practically immune against tuberculosis is calculated to lead to the neglect of the only measures that will eradicate the disease, or keep a herd free from it. The only sound advice to give stockowners on this matter, is to tell them that if they admit a tuberculous subject into their stock, no degree of health or vigour on the part of their animals can be trusted to prevent the disease from spreading; and that, on the other hand, no matter how enfeebled or debilitated any of their animals may be, they will not become tuberculous unless opportunities of infection from tuberculous animals or human beings are permitted.

In conclusion it may be remarked that it is somewhat surprising to observe how little has been done to instruct the general public regarding the nature of tuberculosis, and the principal means by which it is spread among human beings. It is true that the public have often been warned of the dangers of tuberculous meat and milk, but it is open to doubt whether these together account for 1 per cent. of the cases of tuberculosis in the human species. Indeed, it can hardly be denied that this danger has received, and is still receiving, an amount of attention altogether out of proportion to its magnitude. Every reasonable means of limiting the danger of infection from meat or milk of tuberculous animals ought to be sought after, but it is absurd to exaggerate the risk from this source, and to demand the most Draconian measures to guard against it, while leaving full play to the customs which account for 99 per cent. of the cases of human tuberculosis. When the medical profession have come to recognise that very few cases of consumption among their patients are ascribable to infection from the lower animals, and that still fewer cases are congenital, some reduction in the prevalence of the disease may be expected, but no improvement can be looked for as long as practising physicians keep harping on the dangers of meat and milk, and neglect to impress upon the public that contagion from man to man is the great cause of human tuberculosis.

THE ETIOLOGY OF ABORTION IN COWS.

DURING the past few years the older theories of the causation of outbreaks of abortion among cows have to a large extent been abandoned in favour of the view that the condition is contagious. The latter view has been adopted not so much on the strength of direct evidence of its accuracy, as because it appeared to be the only suggested explanation that did not break down when applied to a considerable series of cases. Until comparatively recent times the favourite theory in this country was that which ascribed the act of abortion to what was termed "sympathy." This word, it need hardly be said, was here employed in a special sense, for it would have been too absurd to credit an animal so low in the scale of intelligence as the cow with the feelings that we attempt to describe when we say that we sympathise with a person in pain or distress. Apparently what was intended to be conveyed by the word "sympathy" when offered as an explanation of abortion, was that the sight, or perhaps even the smell, of a cow in the act of abortion might serve as a stimulus sufficient to induce the same act in other cows of the herd. When put in this way the theory was to a certain extent plausible, but it ceases to be that when one reflects that the cow is thereby credited with ability to distinguish between the act of abortion or premature labour, and parturition at full time, for while it is true that when one cow in a herd aborts a series of abortions is apt to follow, a full term parturition in a herd does not lead the other pregnant cows to drop their calves prematurely. It is, however, hardly necessary to show the untenability and insufficiency of "sympathy" as an explanation of abortion, for at the present time it would probably be difficult to find even a stockowner who believes in it.

Ergotised grasses, errors of diet, and sewage contamination of water are, at the present time, the only rival theories of contagion, but it may safely be said that none of these is capable of explaining more than an occasional case of abortion among cows, and it is doubtful whether any one of them is ever accountable for what may be termed an outbreak of abortion. Loose habits of reasoning are apt to lead a person who finds an impure water supply on a farm where abortion prevails to lay the blame on that impurity, and the same illogical method may satisfy him that ergotised grass, or too many turnips, are responsible for outbreaks. But it is not possible to fall into such an error when a series of outbreaks in different districts are submitted to investigation, for nothing is more certain than that abortion is frequently met with in herds where the grass and hay are free from ergot, where the dietary is irreproachable in quantity and quality, and where the water supply is pure.

On the other hand, in investigating outbreaks of abortion, one seldom or never finds that the circumstances exclude the possibility of contagion, and in some cases they point very strongly to that as having been the cause of the outbreak. It must be admitted, however, that the clinical evidence in favour of contagion is very far from overwhelming, and the same may be said of most of the experiments hitherto carried out with the object of settling this question. Much interest attaches in this connection to some experiments which, during the past year, were carried out in Denmark by Professor Bang.[1] In a cow slaughtered while exhibiting the premonitory symptoms of abortion, Bang found a large quantity of thick, yellowish, turbid liquid between the uterus and the fœtal envelopes, and microscopic examination of this liquid showed that it contained, in apparently pure culture, a very small bacterium. Suitable artificial media inoculated from the liquid yielded, after a few days' incubation, an abundant growth of the bacterium. In twenty-one other cases of abortion the same bacteria were found in the uterus or in the afterbirth. Attempts were made to induce abortion experimentally by injecting artificial cultures into the vagina, but in animals that had been thus treated no abnormality was discovered at the *post-mortem* examination thirty days after the culture had been introduced.

Two cows in the third month of pregnancy were then purchased from a herd known to be free from abortion, and on the 14th April 1896, a large quantity of culture of the germ in question was injected into the vagina. The injections were repeated on the 23rd May and 4th June. On the 24th June the first cow gave birth to a five-months-old dead calf, and the bacteria which had been used for infection were found in the exudate present between the uterus and the fœtal envelopes. The other cow was slaughtered on the same day, and in it also the bacilli were found between the chorion and the uterine mucous membrane.

These experiments appear to furnish conclusive evidence that at least some cases of abortion are caused by the multiplication of an organism within the womb, and while they do not absolutely prove that the disease is contagious, they certainly strengthen the clinical evidence pointing in that direction. They also warrant veterinary surgeons in urging stockowners to treat abortion as they would a disease known to be contagious. In this treatment the use of disinfectants ought to play a part, but not the chief part. To arrest the disease reliance must mainly be placed on the measures which have been found most trustworthy in the prophylaxis of the admittedly contagious diseases, that is to say, the strict isolation of every animal suspected of being already diseased. Unfortunately the practitioner is here confronted with a serious difficulty, namely, our present inability to diagnose the disease until the act of abortion is imminent, at which time the cow may already have infected some of her neighbours. But the hope may be indulged that if the disease is due to a bacterium, future investigation will bring into use improved methods of diagnosis.

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[1] Berliner Thierärztliche Wochenschrift, 28th Feb. 1897.

Review.

Points of the Horse: A familiar treatise on equine conformation. By M. Horace Hayes, F.R.C.V.S. Second Edition. London: W. Thacker & Co., 1897.

It is somewhat singular that until the appearance of the first edition of Captain Hayes' book on the subject four years ago, English literature contained no complete work on the external conformation of the horse. That there was a market ready for such a treatise was proved by the demand for the first edition, which was exhausted within six months. The author apparently resisted the temptation to issue as a second edition what would have been merely a reprint of the first, and preferred to spend some time in further study of the subject and in the accumulation of fresh material. On this decision both the author and the public are to be congratulated, for the present work is in several respects superior to its predecessor.

In gauging the merits of Captain Hayes' treatise, one is naturally led to compare it with Goubaux and Barrier's masterly French work on "L'Extérieur du Cheval," which, by the way, an American translation has made available for English readers. In some respects the comparison is favourable to the one, and in some to the other. The English work is greatly superior in respect of its illustrations, of which there are upwards of four hundred, the majority of them being faithful reproductions of photographs. As regards the text of the two books, the difference may be conveyed in a general way by saying that the French authors are more scientific, perhaps in some points more accurate, while the English work treats of things in a more popular, or, as the author would probably prefer to say, a more "practical" manner. But, putting comparisons aside, we may certainly say that Captain Hayes has produced a work that is eminently readable, and one which veterinary surgeons and students will find it worth their while to possess. It will not make every reader of it a good judge of a horse, but it contains information valuable even for those who are already good judges of that animal. In conclusion, it ought to be said that the publishers' share of the work is deserving of the highest commendation.

CLINICAL ARTICLES.

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HORSES POISONED BY SPOILED POTATOES.

By J. M'FADYEAN, Royal Veterinary College, London.

THE following remarkable series of cases of poisoning occurred in the practice of Mr W. Calvert, M.R.C.V.S., Wallingford, and to that gentleman and to the owner of the horses I am indebted for a knowledge of many of the details.

The first animal was taken ill on 9th September while ploughing. The ploughman reported that this horse had appeared to be particularly lively when yoked in the morning, but that about ten o'clock he suddenly fell down as if in a fit. The man got him up again and proceeded to take him home; on the way he fell down twice, but was able to get up again. Just outside the yard gate he fell for a third time, and he never regained his legs, death taking place about one o'clock. The animal in question was a Clydesdale stallion, eight years old. About three o'clock the same afternoon it was reported that a five-years-old gelding belonging to the same stable had been taken ill in the same way, and within two hours the animal was dead. On the following morning (10th September) it was reported that an eighteen-years-old mare in another stable three quarters of a mile distant was in a dying condition, and that two other young horses, aged respectively three years and two years, were showing symptoms. Shortly afterwards a fourth animal, a two-year-old, on the same farm was taken ill, and all four animals died within five hours after the onset of symptoms. Only two horses were left on this second farm, aged respectively four years and seven years, and both died on the following day (11th September), each having been about ten hours ill. On the same day a six-years-old horse, which was a stable companion of the first animal attacked, died with similar symptoms after a four hours' illness, and on the following day (12th September) two other horses, aged respectively three years and four years, in the same stable died after having been ill twenty-four hours.

Except in the Clydesdale stallion, which was reported to have fallen down in a fit, the first symptom noticed in all the animals was weakness and loss of power over the limbs. The animals soon lay down, or fell down, and were unable to get up; while lying down some of them attempted to eat a few mouthfuls, and there was no struggling or other evidence of pain. Towards the last there appeared to be a difficulty in swallowing, and a "fluttering" was noticed in the chest.

In all the animals submitted to *post-mortem* examination the principal organs of the body were found to be healthy, with the exception of the large intestine, the mucous membrane of which showed slight signs of irritation.

The diet of all the horses consisted of steamed potatoes, boiled English barley, old English beans, and old wheat chaff. The quality of the barley, beans, and chaff was unexceptionable, but the potatoes were old, and many of them were mouldy and otherwise much decayed. For some considerable time the potatoes had been stored in sacks, and it is deserving of mention that they were not to any considerable extent sprouted. Some of the horses had been receiving potatoes for ten or twelve days, and others for only three or four days.

As has already been stated, the horses were distributed in two stables three-quarters of a mile apart. In one of the stables all the horses, six in number, were taken ill and died; the other stable contained nine horses, of which five were taken ill and died, while the remaining four were unaffected. Of these four, one, a carriage horse, was fed on dry oats only; two were fed on boiled barley and chaff, the same as was used for the feeding of the other horses, but received no potatoes. The fourth survivor was kept in an open yard, and was a companion of three of the horses that died. In this yard a few potatoes had been left in a feeding rack by a horse which occupied the open yard in the day, and was placed in a box at night. The survivor of this quartette might therefore have obtained a few potatoes, but he was the underling of the lot, and the attendant stated that the others invariably drove him from the rack; it therefore appears not improbable that this animal, like the other three survivors, had not eaten any potatoes. A third stable of farm working horses were fed on dry oats and chaff, and upwards of twenty other horses were running at grass; none of these animals were attacked.

As a chemical analysis made by Mr Bayne, Chemist to the Royal Veterinary College, proved the absence of mineral poison from the stomach contents in one of the fatal cases, and as the circumstances appeared to exonerate all the articles of diet except the potatoes, it was determined to carry out a feeding experiment with them. A quantity of the steamed potatoes left over when the illness broke out was therefore sent to the Royal Veterinary College, and an aged but apparently healthy cab horse was purchased for experiment. Great difficulty was experienced in getting the horse to eat the potatoes, but by keeping him short of other food he was induced to eat nearly two bushels between the 17th and the 26th September. His other diet during this period comprised hay and a little bran mixed with the potatoes. Up till the ninth day of the experiment the horse appeared to be quite well, but on that morning he was found down in his box, and unable to get up. With assistance he managed to regain his legs, but on the same day he fell again, and all attempts to get him up proved fruitless. Death took place quietly thirty-six hours afterwards. The *post-mortem* examination showed that the stomach was healthy, but there were patches of inflammation in the small intestine, and the double colon was inflamed almost throughout, and of a livid tint. A small patch of pneumonia was present in the left lung, but the other organs of the body appeared to be healthy.

A second experiment was conducted at the farm, where a cart horse that appeared to be quite healthy save for disease of his limbs was fed with some of the potatoes. Even greater difficulty than in the first case was encountered in inducing the animal to eat the potatoes. About ten days after he had begun to eat them freely he was found stretched out in his loose-box and unable to get up even with assistance; he died quietly on the following day. It was reported that the *post-mortem* examination showed all the organs healthy with the exception of the large intestine, the lining membrane of which was of a cherry-red colour.

The result of these two experiments, when taken in conjunction with the circumstances previously narrated, leaves little room for doubt that the eleven horses were poisoned by some substance present in the potatoes. As has previously been mentioned, a chemical analysis of the stomach contents in one of the fatal cases proved the absence of any mineral poison, and since the potatoes had been steamed before use it cannot be held that the illness was due to anything of the nature of an infection from organisms present in them. The case appears to be closely related to the so-called "meat poisoning," which has frequently been noted in the human subject after eating unwholesome cooked meat—that is to say, the poison was probably some organic substance generated by the bacteria or fungi which were growing in the decaying potatoes.

In conclusion it ought to be stated that the quantity of potatoes that had been consumed by each of the horses that died varied considerably. Some had consumed two buckets a day for ten or twelve days, and some had only half-a-gallon a day for three or four days. This would appear to show, what on other grounds appears probable, that the poisonous substance was not equally distributed throughout the mass of potatoes, and that it had perhaps been confined to one or two of the sacks.

CLINICAL NOTES.

By J. PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

A DISEASE OF NEW-BORN CALVES.

UNDER this heading the *Journal of Comparative Pathology and Therapeutics* for March 1896 gives an extract from the *Revue Vétérinaire*, which refers to an outbreak of disease of a highly fatal character among new-born calves. Investigations made in the pathological laboratory of the University of Pavia led Monti and Veratti to the following conclusions: *First*, that the disease was a general infection which had its starting-point in the digestive tube. *Second*, this infection, characterised by diarrhœa and albuminuria determined grave alterations in the intestines, the lymphatic glands, kidneys, and brain, and secondarily congestion and small hæmorrhages in a great number of other organs. *Third*, the specific agent was a bacillus related to the *bacterium coli commune*, which passed from the intestines to the circulatory apparatus and led to the development of numerous colonies in all the organs.

In the *Veterinary Record*, 14th April 1894, under the heading of "Scour in Calves" I drew attention to evidence of the dependence of "white scour" on infection, and expressed the opinion that differences between certain conditions included under this name were so marked as to admit of separation into two distinct diseases. Further observation would appear to support this view, and, though for special reasons, bacterial examination was not made, the following account tends to demonstrate that the disease described in the article above referred to exists amongst us and is highly contagious.

The disease in this instance affected the calves in a pure bred Shorthorn herd. Only recently born calves were affected, and in every case death resulted within three days after the onset of symptoms. Every calf born during a period of two months succumbed to the malady, and all the cows went to full time, and were, throughout, in apparently perfect health. The cow sheds were good, and there were no special circumstances in the treatment of the herd which call for remark. It is, perhaps, worthy of note, that the second calf which became the subject of the disease was that of the cow occupying the place next the dam of the first calf which became affected. I observed the same circumstance in the outbreak reported by me in the *Veterinary Record* in 1894. In both cases when the malady broke out there were calves from a month old upwards on the farm, but none of these suffered from this disease. The first symptoms in every case were manifested from twelve to forty-eight hours after birth. The calf was usually noticed lying down, indisposed to rise, often shivering and panting, the coat dry and staring. Soon a violent diarrhœa with tenesmus came on, the evacuations being like muddy water and not extremely fœtid. Shortly the breathing became more disturbed, cough sometimes being observed, and in a few hours the little animal was in a condition of collapse; frequently death occurred in from twenty-four to forty-eight hours after the first symptoms became apparent. The temperature was high, the mercury in one case reaching 107·3° F. I was informed by the attendant that, in some cases, lameness was marked, though the joints were not noticeably swollen.

Post-mortem examination revealed changes similar to those described by Monti and Veratti above mentioned. Gastro-enteritis, nephritis, pharyngitis, congestion of the lungs, and ecchymoses in various parts of the body were most prominent. In all cases that I have examined the contents of the abomasum were like muddy water, sometimes containing a few flakes of curd. Treatment of two affected calves with cinchona, chlorodyne, and peppermint, seemed to have no beneficial effect.

Preventive measures, however, based on the assumption of a contagious nature, have proved highly satisfactory. At first the in-calf cows were removed to a fresh situation, but the calves (as they were born) still became affected. I then advised removal to another place after the lower parts of the limbs, vulva, tails, and lower parts of the cows had been soaked in a solution of sulphate of copper, and the animals allowed to stand on lime. The udders and teats were washed with a solution of boracic acid. This was repeated twice, and afterwards no calves became affected. Strict attention was given as to the disposal of the carcases of the victims of the malady and their excreta, and no cases have since occurred on the farms in which the earlier and the more recent outbreaks were experienced.

Circumstances did not permit of a bacteriological study of the affection. Of the contents of the stomach and intestines of a calf which died in the 1894 outbreak, I gave to a four-weeks-old calf at the College two teaspoonfuls in milk at night, and three teaspoonfuls in milk on the following morning. No symptoms followed this administration. This, of course, may indicate that the virus was not contained in the liquid of the stomach and intestines, or that calves beyond a certain age do not become affected by the digestive canal.

In considering the nature of this malady there is scarcely any means of escaping the conviction that it is infectious. The entrance of the infecting material, both as to point of time and point of entry, are questions to be decided. Certain it is that, if not before or during birth, it must take place very soon after, for sometimes before the patient is more than a day old this disease has laid it prostrate. *Post-mortem* examination has shown me nothing which specially points to infection by the navel, a very vulnerable point in all very young animals, while the investigations of Monte and Veratti lead them to the conclusion that it is by the digestive tube.

An interesting leading article in this Journal for June 1893, on the "Etiology of Diarrhœa in Young Calves," by Jensen of Dorpat, is well worth reading in association with a consideration of the malady under question.

LARGE FIBROUS TUMOUR IN THE UTERUS OF A BITCH.

The subject, an aged collie bitch, was admitted to the College Infirmary on the 16th of November 1896. The owner complained of the swollen state of the abdomen, but otherwise had noticed no deviation from health. The only history to be ascertained was that the abdomen was first noticed to be larger than usual about six weeks previously, and since had gradually increased in size. In the middle of August the bitch was in œstrum, and the owner suspected pregnancy and death of the fœtus. On visual examination the belly was found much enlarged, pendulous, and indeed precisely like that of a bitch far advanced in pregnancy. Manipulation revealed a large hard tumour corresponding in position to the uterus. The swelling was more even than is usual with a uterus containing puppies, though on more than one occasion some irregularity of the surface was apparently noticeable, and gave the idea of being part of a puppy. This was probably some interposed viscus. The general health of the patient appeared in no way affected, though the weight of the abdomen evidently interfered with the comfort of the bitch in walking. On the 4th, 5th, and 6th of December there was a very slight discharge from the vulva, which agglutinated the hair in its neighbourhood. At this time there was an evident indisposition to be interfered with, and the bed was gathered up in a corner after the manner of animals preparing for parturition, but no other indications of the latter condition. There were no signs of ascites, and the firmness of the swelling, etc., discountenanced the view of dropsy of the uterus, while its position and non-interference with digestion, assimilation, or other function of the abdominal organs pointed to enlargement of the uterus. Absence of discharge by the vagina, and of systemic disturbance so long after any possible impregnation, controverted any idea of dead fœtus. In the middle of December the owner was informed that cure was not to be hoped for. Consent for destroying the bitch could, however, not be obtained for a long time, owing to the fact that the owner had heard of a "similar case" having been successfully treated by a great canine specialist, whose remedy he wished to try. He failed to get an opportunity of doing so, and on the 25th of January the animal, with now a tremendously distended belly, but apparently in full health, was poisoned.

Post-mortem examination revealed the presence of a hard dense tumour, weighing 19½ lbs., occupying the whole of the cavity of the uterus. On section this growth was pinkish white, and microscopic examination proved it to be an œdematous fibroma, in which Professor M'Fadyean informs me he found no muscle structure. The other organs were normal.

SUPPURATION IN THE BRAIN OF A HORSE.

For notes on the following case I am indebted to Messrs Prime & Sons, Veterinary Surgeons of Upper Norwood.

The subject was a well-bred chestnut stallion, 14 hands, and rising four years old. On being submitted for examination, it was stated that the animal had recently recovered from strangles, but no evidence of this disease apparently then existed. The first abnormality observed was keratitis with complete opacity of the right cornea, and without any indication of external injury. The animal was said not to have been eating quite satisfactorily for a few days previously, but in high spirits, having been noticed "kicking out" at another horse at 4 P.M. on 18th January 1897. At 10 P.M. on the same day he was found lying down and struggling violently. At 2 A.M. of the 19th, Mr W. H. Prime was sent for. On his arrival the animal was down and struggling. On being got up, he walked in a staggering fashion, with marked loss of control over movements, that of the hind limbs being greatly exaggerated, and after a few minutes the animal fell down in a heap; internal temperature 103°; pulse 60, weak; respirations 18; general surface temperature even, but slight perspiration inside forearms and thighs. Enemata were now used, cold water applied to the head, and morphia and atropine injected hypodermically. At 8 A.M. the same day, the patient, much weaker and quite unable to stand, lay on the right side, the four limbs being moved convulsively, imitating the gallop, with a tendency to work backwards. No coma. Temperature 101° F.; breathing stertorous; power of deglutition lost.

Ice was now applied to the back of the head, but the convulsive movements of the limbs continued to the last. Breathing became more markedly stertorous and the respiration more frequent. The eyes were rolling convulsively to and fro, but corneal reflex remained, and there was no evidence of coma before death, which took place about 2 P.M. on the 19th.

Post-mortem examination revealed a healthy condition of the internal organs, except the spleen, which was hypertrophied, weighing a little over 9 lbs.

On removing the skin from the head, no ecchymoses, fractures, or other evidence of external injury were appreciable. The cerebral meninges showed no sign of inflammation, and there was no collection of fluid or pus in the arachnoid spaces. The veins of the brain were engorged. The cavities of the olfactory bulbs and the lateral ventricles (right and left) were nearly filled with thick white pus. There was no evidence of an abscess having existed in the cerebrum. The Schneiderian membrane appeared normal, and the cranial sinuses contained no pus.

An examination of the partially dissected brain sent me by Messrs Prime showed the presence of streptococci. This leaves little doubt that the suppuration in the cerebral ventricles and the consequent symptoms were due to the specific microbe of strangles, which, it is well known, occasionally occupies this situation.

TUMOUR IN THE BRAIN OF A DOG.

Tumours associated with the plexus choroides, in the lateral ventricles of the brain of the horse, are of comparatively common occurrence. The instance to which I now refer is the first of its kind coming under my own notice, and in the several text-books which I have searched I find no reference to the existence of tumours in the same situation in the brain of the dog. From the large number of examinations made of the brains of dogs compared with those made of the brains of horses, and no mention being made of cerebral tumour, it is probably a rare condition.

The growth in the case now under notice was found in the right lateral ventricle, attached only to the vessels of the plexus choroides. In size about that of a walnut, being thickest posteriorly. Professor M'Fadyean, who has made a microscopic examination of a section, reports that the growth is an adenoma.

The right lateral ventricle contained a considerable quantity of straw-coloured fluid.

Both kidneys gave evidence of chronic nephritis. In the spleen there was a small growth apparently of the nature commonly observed in this organ of the dog. Unfortunately, however, this was mislaid and was not forthcoming until too late for microscopic examination. No other abnormality was observed at the autopsy.

The subject of the above, an aged brindled bull-dog, was brought to the College Infirmary with a history of gradual wasting of flesh and increasing dulness. Up to a fortnight before this, the dog was not noticed at all different from usual. In fact, previously he had enjoyed splendid health. The appetite was good up to the time of admission. There had been no symptoms of paralysis or exaggerated movements. Within the past few days the dog seemed a little near-sighted.

On being examined before admission, emaciation was well marked, temperature normal, and pulse slow but regular; the animal, though apparently capable of every movement, walked listlessly, and with evidence of great debility. There was a strange, dull facial expression, and the pupils were dilated. In the kennel the sitting posture was generally assumed, the head and nose being directed to the right. As far as could be judged the animal could see only fairly well, but the hearing was more evidently impaired, and he was not easily aroused by calling. On being offered food, however, the patient readily came for and ate it. During his stay here the appetite was good, and there was no difficulty in swallowing. In the intervals between feeding times, the dog remained usually in one position in a listless state.

The general impression conveyed to the mind of the observer was that of "silliness" of the patient. The peculiarity, evidently connected with cerebral disturbance, suggested the possibility of rabies, and the animal was accordingly placed in a specially secure position for observation. After a few days, during which there were no developments indicating rabies, chronic cerebral disease, probably tumour, was diagnosed, and the animal poisoned, *post-mortem* examination yielding the results before described. Had the animal been allowed to live, it is possible that exaggerated and erratic movements might have showed themselves, as is not uncommon in the horse with cerebral tumour, when these in conjunction with the strange behaviour of the dog in the intervals might have led to the conclusion that he was a subject of rabies.

CONTAGIOUS PLEURO-PNEUMONIA OF HORSES.

This affection, which for many years has been more or less commonly met with among our horses, was apt in former times to be confounded with influenza, and even at present is too often included in the term. During the last fifty years contagious pleuro-pneumonia has occasionally shown itself in a very virulent and enzootic form, and its dissemination by newly purchased animals has long been recognised. It is a view largely accepted, that on fresh importation of contagious disease from a foreign country, its manifestations are usually severe. Recent severe outbreaks have, I believe, with some show of truth, been credited to horses imported from America and Canada. It is certain that in the fall of 1896, when the weather became boisterous and colder, and the passage across the Atlantic rough and protracted, many freshly imported animals were affected and were the means of spreading this highly contagious and destructive malady amongst our horses.

The following general remarks, though based on a more extended experience with the affection, are particularly suggested by two outbreaks in London stables, occurring within the past three months. The object in view will, however, probably be best served by specifically referring to four cases under my care during parts of the last and present months. These are certainly typical of the cases with which I have met.

Of the introduction of the disease by Canadian horses in one of the outbreaks there can be no doubt.

The owner of the animals affected in the other outbreak attributes the infection to Canadian horses which stood for some time in one of his stables, but evidence of this source of infection is open to question, indeed, is not supported by facts, for four affected animals have come under my treatment which have never entered the stable supposed to have been contaminated by the "Colonials." From an analysis of the circumstances, I am disposed to think that railway horse-boxes are not without the pale of suspicion, and that the centre at which subsequent contamination took place is a stable to which all this owner's horses are taken on coming off the rail, and where they remain some time before proceeding to their permanent destination. It is from this stable that the four animals whose cases I am about to describe were brought to the College Infirmary. It may be well to mention the fact that since Christmas this owner has had twenty-three animals affected, out of which eleven have died. I had opportunity of seeing some of these cases in their own stables, and of gaining full information from the veterinary surgeon who was in constant attendance, and there can be no doubt as to the identity of the disease affecting the horses at the owner's residence and the animals admitted to the College, which had never been there.

On 23rd February a bay cart mare and a bay cart gelding were sent to the College. On admission both animals appeared vigorous, though perhaps a trifle dull. The respiration was much disturbed, the movement of ribs and flanks being excessive. The temperature of the mare was 106·2 and that of the gelding 105·4. The pulse, respectively 60 and 70, was strong and regular.

Auscultation and percussion revealed some evidence of abnormality in the right lung of each animal, but gave no definite idea except that in parts the normal sound was interrupted. There was no discharge from eyes or nostrils, no suffusion of the eyelids, no swelling or stiffness of limbs, and the conjunctivæ were a little hyperæmic, their colour a reddish-brown.

Each animal was placed in a large loose-box, rugged, bandaged, and given an antipyretic draught containing 3 ounces each of compound tincture of cinchona and rectified spirit, 1 ounce of spirit of nitrous æther, and 4 ounces of solution of acetate of ammonia with water. A small allowance of oats, bran, and chaff was then offered, and this being greedily eaten, a fair feed (including hay) for a healthy horse was given. By the morning this had all been eaten.

It is probable that during the very short invasion stage some inappetence may be observed, indeed I think it often happens that a meal or two may not be cleaned up. After this, however, the maintenance of the appetite is to me a very common, perhaps not constant, but certainly remarkable clinical feature of this disease which has not been generally recorded; indeed, I find in our most credited English, French, and German text-books expressions which, when the foreign are translated, are identical—"The appetite is almost entirely suppressed." It may, in this light, be interesting to give the clinical table of four cases, two of which proved fatal, and the others ended favourably.

Bay Gelding, No. 1.

	Temp.	*Pulse.*	*Resp.*	
Feb. 23	105·4	60	30	Feeding well; eats a full allowance.
„ 24	106·2	60	20	Do. do. do. do.
„ 25	105·4	50	20	Do. do. do. do.
„ 26	107·1	72	25	Do. do. do. do.
„ 27	106·6	72	28	Do. do. do. do.
„ 28	—	—	—	Do. do. do. do.
Mar. 1	106·6	72	30	Do. do. do. do.
„ 2	106·2	75	36	Feeding not so well; part of food left in manger.
„ 3	106·2	90	36	Not feeding.
„ 4	105·8	80	40	Not feeding.
„ 5	Died early in the morning.			

Bay Mare, No. 2.

	Temp.	*Pulse.*	*Resp.*	
Feb. 23	106·2	70	45	Feeds fairly; left a small quantity.
„ 24	105·8	75	46	Feeds well; eats full allowance.
„ 25	105·4	72	48	Do. do. do. do.
„ 26	105·4	72	45	Do. do. do. do.
„ 27	105·2	75	46	Do. do. do. do.
„ 28	—	—	—	Do. do. do. do.
Mar. 1	104·6	72	40	Do. do. do. do.
„ 2	104·2	72	36	Do. do. do. do.
„ 3	103·6	75	30	Do. do. do. do.
„ 4	104·2	80	40	Do. do. do. do.
„ 5	104·6	72	35	Do. do. do. do.
„ 6	104·2	72	30	Do. do. do. do.
„ 7	—	—	—	Do. do. do. do.
„ 8	104·2	68	28	Do. do. do. do.
„ 9	103·8	66	24	Do. do. do. do.
„ 10	103·2	64	24	Do. do. do. do.
„ 11	103·2	63	24	Do. do. do. do.
„ 12	104·8	68	40	Feeds not so well; only eaten half allowance.
„ 13	106·1 Died.	90	60	Appetite entirely suppressed.

Black Gelding, No. 3.

	Temp.	*Pulse.*	*Resp.*	
Feb. 26	104·4	64	28	Feeds well
„ 27	101·0	48	14	„
Mar. 1	104·2	56	20	„
„ 2	102·8	50	20	„
„ 3	101·8	48	19	„
„ 4	102·2	46	14	„
„ 5	103·0	48	15	„
„ 6	103·4	56	18	„
„ 8	100·8	48	15	„
„ 9	100·6	47	13	„
„ 10	100·4	46	12	„

Remained normal till 14th, when the patient was discharged convalescent.

Chestnut Gelding, No. 4.

Temp.	*Pulse.*	*Resp.*	
104·8	52	60	Feeds well
105·0	64	28	„
104·8	64	38	Not quite cleared up food
101·0	48	20	Feeds well
101·0	46	19	„
101·0	48	18	„
100·6	46	15	„
100·0	47	15	„
100·0	46	14	„
99·8	45	13	„
99·8	44	12	„

Discharged convalescent.

Recurring to Cases Nos. 1 and 2, which ended fatally. No. 1, the bay-gelding, continued, to general observation, in much the same condition for three days after admission, when, coincident with the rise of temperature to 107·1° on 26th February, some signs of pleurisy developed, the thoracic movements became restricted, and loss of vesicular murmur and increased tubal sound with the increased number of respirations pointed to consolidation or collapse of lung tissue. Still, though not quite so lively as on admission, there was no loss of appetite till 2nd March, nor any striking debility or depression until 3rd March, eight days from the date of the first observation, when the animal lay down and showed some slight signs of abdominal pain, and the appetite was completely suppressed. From this time to the morning of the 5th, when the animal died, dyspnœa was marked and abdominal uneasiness occasionally manifested.

Post-mortem examination revealed extensive disease of the ordinary character in the lungs, pleura and thorax. The lungs on section had in parts a marbled appearance, similating that of contagious pleuro-pneumonia of cattle. The peritoneum and abdominal viscera showed marked change, and the large intestines were intensely inflamed as a result of the attack of the embryos of sclerostoma tetracanthum, a condition that has not infrequently been observed as a complication in this and other fatal affections, and one so common that it can scarcely be left out of consideration whenever serious abdominal disturbance ensues in course of any disease. How far they may prove a predisposing cause of general infection or blood-poisoning, by providing ports of entrance for micro-organisms and their products, it is impossible to estimate.

The Bay Mare, No. 2.—The course of this case for a fortnight was much the same as that of No. 1 for its first three days. Cough was more frequent, and over the right lung there was a considerable area of dulness. On 8th March there was stronger evidence of pleurisy, which continued till death took place on the 14th. During the whole course of the disease this animal did not lie down, and until the 8th showed no indisposition to move about the box. No grunting or other evidences of pain on pressure of the chest wall. There was only occasionally a small amount of discharge from the nostrils, and the breath was not noticed to be fœtid.

Post-mortem examination revealed some collapse and congestion of the lungs, and pleurisy of the ordinary fibrinous character and a very fœtid odour.

Of the two animals which recovered (Nos. 3 and 4) it is only to add that on admission and for four days afterwards the conditions were not distinguishable, except by the record of temperature, etc., given above, from those which died. On the fourth day there was noticeable an increase in the amount of urine and fæces, and up to this time there had been loss of vesicular murmur and increased dulness over the right lung, which soon after began to clear up rapidly, and the animal was discharged convalescent about three weeks after admission.

The general and medicinal treatment of the patients was the same in each case. Febrifuge draughts of composition similar to that named above, inhalations of steam medicated with eucalyptus, and small doses of sulphate of magnesia, were administered for the first four days.

Then or before, if giving draughts appreciably disturbed the animal, balls containing 2 drachms each of quinine and iodide of potassium in bulk were substituted.

Some of the more prominent features of this affection appear to me to be, its highly contagious nature; that imported animals are most liable to be the means of introducing the virulent form; that only a brief sojourn in an infected place or with affected or incompletely recovered animals is necessary to contract the disease; that the invasion stage is very short; that the nervous prostration, cardiac debility, and consequent phenomena, typical of influenza, are absent; and that, if the ordinary pneumonia and pleurisy of the horse are not contagious, there are yet no means independent of the history of distinguishing in the early stages between it and the contagious. Treatment with ordinary antipyretic medicine appears to be of little avail. The affection is due to a specific micro-organism, and I fear that until some specific material which may accrue to culture or serum pathology investigation, or otherwise, is obtained, we shall have to depend on the ordinary rules for preventing such contagious diseases. There seems to me plenty of room for the energies of the Animals' Division of the Board of Agriculture as well as private enterprise in this direction.

NOTES ON SOME NEW DRUGS

By FREDERICK HOBDAY, Professor of Therapeutics, Royal Veterinary College, London.

EUCAINE HYDROCHLORATE; A COMPARISON BETWEEN THE ACTION OF THIS DRUG AND COCAINE HYDROCHLORATE ON THE HORSE, DOG, AND CAT.

BEFORE operating upon a part it is often desirable to use a local anæsthetic, and in connection with this agent it is absolutely necessary that the operator should choose not only a reliable one, but also one that can be used with safety. It is well known in veterinary practice that the use of cocaine for operations upon the dog and cat must be attended with the greatest caution, as the toxic doses are very small; when, therefore, another agent is produced, said to possess the same anæsthetic effect without incurring the same danger, it must of necessity be welcomed and given a fair trial. 'Eucaine; a synthetic product having the same formula as cocaine, is a new drug recently introduced to the notice of the medical world, and said to possess the above qualities; up to the present, opinions given upon its value appear to vary very widely. For example, Professor Charteris and Dr M'Lennan,[1] in a communication recently made to the Royal Society of Edinburgh, speak very favourably of its local anæsthetic effects, especially for ophthalmic and dental operations, and state that its use subcutaneously is attended with much less danger than cocaine.

Dr G. W. Spencer[2] publishes in an American journal his opinion that it produces the most complete anæsthesia of any local anæsthetic up to date, and that its effect is rapid and safe, producing no after effects; that compared with cocaine the anæsthesia is as good, and the toxicity less. An advertisement published in the *Therapist*[3] states that the patient can tolerate, if required, an injection of 30 grains eucaine without injury.

On the other hand Professor Ponchet,[4] in a paper read before the Société de Thérapeutique, 27th January, asserts that eucaine is a powerful cardiac depressant, and in some instances produces sudden death without warning. He regards eucaine of more danger and less use clinically than cocaine. Also in a review upon eucaine, the *Lancet*[4] states that "whether it deserves all the praise as to its greater safety than cocaine is open to doubt."

In the College Clinique we have now used the drug in some forty cases, some of them being especially favourable for making a comparison between the action of eucaine and cocaine.

The results have been to confirm the opinion that the toxic dose of eucaine is somewhat larger than that of cocaine, and at the same time that for operations on the cornea of the eye eucaine compares very favourably with cocaine, although anæsthesia is not produced quite so rapidly; but for its local anæsthetic effects when injected subcutaneously or applied locally to places other than the eye, the results obtained in the horse, dog, and cat, have not by any means come up to the results obtained with cocaine. We have found, however, that a mixture of cocaine and eucaine dissolved in water possesses the better anæsthetic properties of the cocaine solution, and can be tolerated in larger dose than cocaine alone; this is undoubtedly of advantage when removing large tumours from animals in which the toxic dose is so small.

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[1] Veterinary Journal, March 1897.
[2] British Medical Journal, 20th February 1897.
[3] The Therapist, 15th October 1896.
[4] Lancet, 27th February 1897

By way of illustrating the above remarks the following cases may be quoted.

Case I.—A bull-dog had a solution of eucaine (2 grains to 1 drachm) painted on the cornea and conjunctiva of the left eye with a camel hair brush for one minute, the right eye being similarly served with a solution of cocaine of the same strength; the result was a satisfactory and complete anæsthesia of the cornea and conjunctiva with the cocaine in one minute, with the eucaine in three minutes.

Case II.—A cat, served similarly, showed complete anæsthesia of the cocaine cornea in one minute, and of the eucaine cornea in two minutes, the latter being distinctly sensitive one minute after the commencement; complete anæsthesia was still present in each cornea twenty-five minutes later.

Case III.—A bull-bitch, with ulcerative papillomata on each side of the vulva. The tumours on the left side were painted continuously for five minutes with a watery solution of eucaine (2 grains in ʒi.), and those on the right side for four minutes with a solution of cocaine of the same strength; when the operation was commenced three minutes later, the eucaine side being taken away first, the animal evinced a considerable amount of pain, whereas on the cocaine side no pain was expressed, anæsthesia being well marked.

Case IV.—A bull-terrier, with everted rectum necessitating amputation; the parts were painted thoroughly and continuously for five minutes with a solution of eucaine (4 grains to a drachm of warm water), and attempts to operate were made at intervals during the next ten minutes, but as the animal at each time evinced pain, the amputation was performed under chloroform. A fortnight later the same animal everted a further portion of rectum, necessitating a second operation of exactly the same character as the last; the part was painted with a solution of cocaine (2 grains to a drachm of water), and in four minutes was quite insensitive, the operation being performed without the slightest manifestation of pain. Some ten days later a still further portion was everted, this being amputated under the influence of a grain of cocaine and a grain of eucaine dissolved in a drachm of water, a very satisfactory anæsthesia being produced after painting the parts for five minutes.

Cases V. and VI. were cats, suffering from hæmatoma of the ear. A solution of eucaine (2 grains in ʒii.) was applied continuously to the under surface of the ears, where quite devoid of hair, in one case for eight minutes, in the other for nine minutes, but without producing any local anæsthetic effects. This is an operation in which we frequently use cocaine with beneficial results.

CASE VII.—A horse, suffering from two large elbow tumours, received subcutaneously in different places around the tumours on the left elbow 8 grains of eucaine dissolved in 4 drachms of water. The operation was commenced ten minutes later, but the animal evinced pain even directly over where the eucaine had been injected; the other elbow tumour was removed without any anæsthetic, the result, as regards manifestation of pain, being apparently exactly the same in each case.

CASE VIII. was similar to Case VII., with the exception that only one elbow tumour was present, and the horse received 10 grains of eucaine in 2½ drachms of water. During the operation the animal evinced slight pain at intervals, the anæthesia certainly not being as complete as we have on other occasions got from similar solutions of cocaine, although we did not get the same hyperæsthesia and clonic spasms of the muscle of the legs as would be produced by a similar dose of cocaine.

CASE IX.—A dog of delicate breed, the eyelid being operated upon for entropion, had a solution of half-a-grain of eucaine dissolved in half-a-drachm of water painted on the eyelid for three minutes. The operation was commenced two minutes later and performed almost painlessly, slight pain only being expressed when the sutures were inserted.

CASES X. and XI.—In each case a kitten, for excision of eyeball, had the parts painted for three minutes with a solution eucaine (1 grain in 40 minims of water), the operation being commenced four minutes later. Anæsthesia seemed complete in the superficial parts which the eucaine had reached, but not when the deeper parts were severed.

CASES XII. and XIII. were dogs in which tumours were to be removed. One (a collie) received 2 grains of eucaine dissolved in 40 minims of water subcutaneously in four places; after a lapse of five minutes the operation was commenced and the tumour removed without any sign of pain being manifested. The other, a St. Bernard, received 2 grains dissolved in a drachm of warm water in six places around the tumour subcutaneously; five minutes later the operation was commenced and continued for seven minutes, no sign of pain being evinced.

CASE XIV. was a cat for castration. After removal of hair the scrotum was painted thoroughly for two minutes with a solution of half-a-grain of cocaine and half-a-grain of eucaine in 1 drachm of water. The animal evinced no pain when the scrotum was incised two minutes later; the testicle and spermatic cord were then painted with the solution and incised, no pain being expressed.

CASE XV.—In order to produce local anæsthesia for a minor operation on a cat weighing 7 pounds, a mixture of half-a-grain of cocaine and half-a-grain of eucaine dissolved in 1 drachm of water were injected subcutaneously. Perfect local anæsthesia was obtained in two minutes, the animal evincing no toxic symptoms, whereas had 1 grain of cocaine alone been used the result (inferred from previous experiences) would certainly have been death.

CASE XVI.—A cat for removal of a large mammary tumour; this was at first painted with a solution of eucaine (1 grain to 1 drachm) for five minutes, the local anæsthesia produced being only slight.

A solution of cocaine and eucaine (half-a-grain of each to 1 drachm of water) was then used, an excellent anæsthesia being obtained in three minutes.

In regard to the amount of eucaine that can be used subcutaneously with safety, and a comparison between the toxic symptoms of cocaine and eucaine, the limit of safety in the case of the cat and dog is about one-fifth of a grain to each pound of body weight; in the horse there is little likelihood of a case arising in which more than 20 grains will be necessary to be given. The most I have seen administered at one time has been 1 drachm dissolved in 2 ounces of warm water, the same animal receiving a few days later a similar dose of cocaine. The difference between the symptoms was well marked; in the case of the overdose of eucaine the symptoms were as follows. After five minutes, local anæsthesia at the seat of injection, but not extending so far around as in the case of the cocaine; after ten minutes slight uneasiness and distinct hyperæsthesia, but nothing like that produced by even a 10 grain dose of cocaine; after fifteen minutes involuntary nodding movements of the head, animal standing quite still and when made to move doing so very cautiously. Up to this stage the pulse and temperature had remained unaltered (the former 36 and regular, the latter 98·5°). Twenty minutes after the injection the head was nodding more frequently, the body swaying backwards and forwards, and a movement as of gulping or swallowing taking place about six times per minute. After forty minutes the nodding movements had almost entirely ceased, and the animal stood very quiet; general hyperæsthesia and local anæsthesia still well marked; the pupil of the eye about the same as at the commencement. One hour and twenty minutes after the injection there was nothing abnormal noticeable; hyperæsthesia had apparently all passed off, local anæsthesia was still present but not to such an extent as at first; animal walked all right; temperature was 98·4° and pulse 32. No further effects were manifested, the animal eating a hearty meal about nine hours later and continuing apparently well.

With the same dose of cocaine four days later the symptoms presented were very different. At the commencement the pulse was 48 and temperature 99·8°. In three minutes local anæsthesia was well marked, respirations accelerated, great hyperæsthesia, the ears being pricked up and head nodding occasionally. In six minutes well marked twitchings of the muscles of the limbs, animal standing in one place, snorting at intervals, pupil of eye distinctly dilated.

After twenty minutes animal very restless and excited, not able to keep legs still at all, picking up first one leg and then another, salivating, snorting, respirations accelerated, had passed fæces, very great hyperæsthesia, pupil much dilated; the temperature was now 101·6°, pulse could not be obtained on account of the restlessness.

After thirty minutes the local anæsthesia was perceptibly passing off. After fifty minutes the symptoms were perceptibly moderated. After three hours there was still hyperæsthesia, slight rigors of muscles of limbs, no signs of local anæsthesia, respirations still somewhat accelerated, body sweating in patches, and animal had passed fæces several times since last time of marking observations; temperature 102·8°. Animal took food readily. When observed again two hours later all symptoms had completely disappeared, and no further abnormality was noticed. Appetite continued good during the succeeding days.

In the smaller animals 4 grains produced toxic symptoms in a dog of 12 pounds weight, but the animal eventually recovered, whereas such a dose of cocaine would certainly have produced death. Again, a dog 35 pounds weight received subcutaneously 8 grains without producing any very noticeable symptoms. In a cat in which local anæsthesia was attempted by 1 grain of eucaine in solution no toxic symptoms followed, but by a careless mistake a couple of days later, a grain of cocaine produced in the same animal toxic symptoms and death.

The toxic symptoms are not noticeable so early as those of cocaine; hyperæsthesia is present but not to such an extent; the rectal temperature rises, salivation takes place but not so thin and watery as with cocaine, and gulping movements are not so well marked. Similar clonic involuntary spasms take place as with cocaine, but at much longer intervals, and the animal becomes prostrate; respiration is accelerated; in the cat the pupil certainly becomes widely dilated; consciousness is not lost until shortly before death, and there is not the same peculiar quietness and absence from moaning which is so characteristic of cocaine. In one case death appeared to occur from cardiac failure, as there were a few distinct respiratory efforts after the heart had ceased. *Post-mortem* appearances show nothing very diagnostic; if examination take place within a short time after death the back of the pharynx, œsophagus, and in some cases, the stomach as well, are found full of frothy mucus, but if the *post-mortem* examination be delayed some hours then even this will have disappeared.

EXTRACT HAPLOPAPPUS BAYLAHUEN LIQ.

About two years ago I received a sample of the above drug from Messrs Christy & Co. for trial in veterinary practice. Since then it has been constantly in use for certain classes of cases, and I think that the results justify its taking up a permanent position amongst our therapeutic agents. According to the description forwarded by Messrs Christy, "the extract is a preparation obtained from the leaves and stem of the Haplopappus Baylahuen Remy, a plant belonging to the Aster family, of the natural order compositæ; this plant is indigenous to the cordilleras of the Chilian province Coquimbo, and has been described by Remy. The stem is woody, and the leathery leaves are somewhat crowded together; the latter are distinctly serrated, especially at the apex, and at their base are somewhat sheathed.

"There is a yellow, strong-smelling, viscid substance, generally secreted by hair-like glands, and covering both the leaves and most parts of the stem, causing it to appear as if dipped in some resinous mass; it is this latter which is considered to contain the agents which give it a therapeutic value. In Chili the drug is used internally in cases of weak digestion as a stimulant; and as an emmenagogue.

"According to Baille, the solution in 150 parts of water may be used as a splendid remedy for diarrhœa; it is also useful in cases of acute and chronic dysentery. Externally it is used for healing the wounds of animals. We have never heard that it possesses any toxic property."

Having at that time quite a number of cases of diarrhœa in the dog and cat under treatment, I determined to give it a trial; the results were very satisfactory in cases in which the diarrhœa was traced to the presence of some irritant in the intestinal tract; in some cases treatment was preceded by a dose of castor oil; in others the Baylahuen was given immediately; in all, the appetite improved from the commencement of the administration, and in no case was the dose followed by symptoms of vomiting or nausea.

The usual method adopted was to give a dose of castor oil on the first day, and follow this up by doses of Extr. Baylahuen three or four times daily; in the majority a cure was effected on the third or fourth day.

As a stomachic we administered the extract in water two or three times daily with beneficial results. Externally for wounds on my own hands, on the bodies of the horse, dog, and cat, a proportion of 1 to 16 or 20 of water gave results very similar to those which follow the application of Tinct. Benzoin Co., but not quite so irritating on the first application, the styptic action being about the same as that possessed by this drug.

In three cases of wounded orbit, after excision of the eyeball, the results of cleansing and dressing three times daily with a 1 in 20 solution were especially good. Altogether in the dog and cat observations were made on about fifty cases, and in the horse on ten cases, the dosage being for the cat from 10 to 30 minims, for the dog from 20 minims to 2 drachms, and for the horse from half-an-ounce to an ounce and a half, given two or three times daily, either in water or flour gruel. Its internal therapeutic value was shown to be that of a stomachic and astringent. As a parasiticide it is of no value, some intestinal worms from the ox being immersed in the pure extract for five minutes by Professor M'Fadyean, without being killed or indeed much disturbed. Being desirous of testing its results in cattle, especially calves, I obtained the collaboration of Mr Freer of Uppingham, Mr Storrar of Abergavenny, and Mr Scott of Bridgewater, who each very kindly consented to give the new drug a trial.

Mr Freer used it in foals and calves suffering from diarrhœa, in half-ounce doses, with marked beneficial effect in most instances.

Mr Storrar reports on its use in lambs, foals, and calves, in 1 drachm doses with very satisfactory results, several cases of severe diarrhœa yielding to treatment after only three or four doses, and he concludes by expressing the opinion that it is a very safe and effective astringent tonic, free from the depressant effects produced by some of the usual sedatives made use of in diarrhœa.

Mr Scott speaks very highly of its intestinal astringent and stomachic properties in cows, calves, and lambs, and states that the results obtained far exceeded his expectations, cures being generally speedily effected without any nauseating or other ill effects. Mr Scott also observed that better results followed small and frequently repeated doses than large doses with long intervals between. Cases were tried with and without the previous administration of oleaginous purgatives, the inference drawn being that cures were more rapidly effected when the oil was given, although the latter was not absolutely necessary.

The doses used were: for cattle from half-an-ounce to 1 ounce, for calves 1 drachm to 2 drachms, and for lambs from 15 minims to 1 drachm.

That these doses can be increased if desired the following observations, which were made in order to test the toxic property, will show.

Mr Scott gave a dog, 42 pounds weight, nine months old, 2 drachms of solid extract of Baylahuen after fasting the animal for twenty-four hours (this solid extract was prepared in order that it might be readily administered in the form of pill or ball, and was about four times as strong as the fluid extract). Two hours later 3 drachms more were given, and after another two hours a further dose of 3 drachms; the animal thus got 1 ounce within about four hours. The only symptoms produced were those of mild alcoholic intoxication, viz., sleepiness, dulness, and unsteadiness of gait, but the animal seemed quite bright and responded when spoken to. Two hours after the administration of each dose all effects appeared to have passed off, and there was nothing abnormal noticeable.

In using the fluid extract, I gave a dog, weighing 12 pounds, 1 ounce mixed with 1 ounce of water; another dog weighing 35 pounds got 2 ounces mixed with 2 ounces of water, and a full-grown sheep got 4 ounces mixed with 4 ounces of water; at no time afterwards was any untoward or objectionable symptoms noticed, the animals taking their food regularly and appearing bright and lively.

In summing up the results there certainly seems excellent reason for placing Extract of Baylahuen amongst the therapeutic agents at present made use of as stomachics and astringents in the treatment of diarrhœa, especially of young animals; its particular advantage over opium and some of the other agents at present in use being that it is free from all nauseating or after-depressant effects, and does not appear to possess any toxic property when given in anything like reasonable doses.

A CASE OF GLANDULAR ENLARGEMENT IN A MARE, SIMULATING TUBERCULOSIS.

By Sydney L. Stephens, M.R.C.V.S., Wadebridge, Cornwall.

The subject was an aged grey Shire mare, the property of a gentleman farmer, who had bought her in the early part of last summer as a mare in foal and in good condition, from a stud farm in the Midlands. On arrival at her new home, some three months later, she was in very poor condition, but seemed hearty, and worked well on a liberal diet.

On 30th November, whilst at work, the mare was seized with violent colic, went to ground gasping for breath, and was with difficulty got into her loose-box. She was quietened by the administration of a colic drench, followed two hours later by a purgative ball. The heart sounds were weak, and undue pressure within the thorax evident. By 2nd December a hard swelling of the size and shape of a bun had appeared bilaterally on the third and fourth cervical vertebræ, and there was also some subcutaneous effusion under the body. Diuretics and tonics prescribed. Tuberculosis or melanosis feared. On 5th December the mare, on being exercised for the first time since the attack, repeated the colicky performance of 30th November. On the evening of 15th December she was inoculated with tuberculin, and her temperature was regularly recorded until 7 A.M. on the 17th. There was no reaction. On 18th and 22nd December exercise was again attempted, and with the same results as before. Her appetite remained unimpaired during the whole period, until she was found dead on the morning of the 17th January. There was no evidence of any death struggle.

The autopsy next day revealed an emaciated carcase, with former swellings almost disappeared, but a soft enlargement inside the off thigh, involving the mammary gland, and containing a serous fluid. There was no fœtus present. The abdominal cavity contained about 1½ gallons of serous fluid. Peritoneum much discoloured. Mesenteric blood-vessels engorged. Mesenteric glands greatly enlarged and softened. Mucous membrane of stomach and intestines apparently healthy. Spleen enlarged, but free from tumours. Liver darker than normal, and very friable. Kidneys normal. Thorax contained about 2½ gallons of serous fluid. Lungs pleural and pericardium discoloured. Heart hypertrophied. Tracheal glands tremendously enlarged.

Portions of the viscera were forwarded to Professor M'Fadyean, who reported that the enlarged mesenteric and tracheal lymphatic glands had a structure which indicated a condition of hyperplasia, the bulk of the new tissue being composed of uni-nuclear lymphocytes. There was no evidence of caseation or other degeneration, and no giant cells were present. Sections and scrapings from the diseased glands were stained for tubercle bacilli, but with a negative result.

CYSTIC CALCULI IN A BITCH.

By HENRY GRAY, M.R.C.V.S., London.

SUBJECT.—An aged (about ten years) small fox-terrier bitch, very fat. For some time she had been noticed to have a great difficulty in passing urine, the act appearing to cause her pain. The last two days before I was consulted she made several attempts to pass urine, which was bloody and in small quantity; there was also occasional vomiting, drowsiness, loss of spirit, a desire for sleep, and no inclination for exercise. On 20th March she was brought to me, because the owner thought she had been injured. On examination I found strings of bloody fibrin hanging from the inferior commissure of the vulva. The vagina was constricted, as it generally is in old bitches which have never borne young, and on separating the lips of the vulva a large quantity of bloody urine escaped. About 1½ in. from the vulva I detected a calculus the size of a large marble in the urethra, and on manipulating it with the two index fingers—one in the rectum and the other in the vagina—seven small calculi, each about the size of a tare and of a triangular form, shot from the vagina when the bitch made some expulsive efforts. After several unsuccessful attempts to dislodge the large calculus I chloroformed the animal, dilated the vagina by means of a Kramer's ear speculum, and made a vertical incision on to the stone and extracted it. Further search was made to find if there were any more calculi in the bladder, but in this direction I failed. However, on making a *post-mortem* examination on the bitch, which died during the early hours of the 21st March, I found two other calculi—one the size of a marble, which had since the operation become lodged in the neck of the bladder, and the other the size of a tare seed. The mucous membrane of the bladder was the seat of intense inflammation, its surface being rough and of a dark almost hæmorrhagic tint. The gastric mucous membrane was much congested.

Although I have frequently seen urethral calculi in the dog, the above-mentioned case is the first one I have encountered in the bitch. In the dog my experience leads me to conclude that whenever there is hæmaturia accompanied with calculi the case ends fatally. This may be due, perhaps, to the fact that I have only seen cases which had been neglected a day or two before I have seen them.

Report.

TICK FEVER IN QUEENSLAND.[1]

The observations and experiments recorded in this report were made by Mr W. Collins and Dr J. Sidney Hunt, at the instance of Mr P. R. Gordon, Chief Inspector of Stock, acting under the authority of the Hon. Horace Tozer, Home Secretary.

The disease to which they refer had been known in the Northern Territory of South Australia for a considerable number of years, without, however, apparently attracting the serious attention of the authorities of that colony. Thence it had been carried across the Queensland border, and by the end of the year 1894 had already spread widely in the Gulf district. In 1894 the trouble had become so serious that Mr C. J. Pound, the Government Bacteriologist, was sent to investigate and report upon the matter. During 1895 the disease had spread South and East with alarming rapidity, and before the beginning of 1896 had extended to the Eastern coast, where its ravages were so peculiarly destructive as to give rise in certain places to the idea that it was a wholly different and more malignant disease than that which had been reported from the Gulf country. In consequence of representations to this effect a commissioner was sent by the Government to inquire into and report upon this question, with the result that the disease was found to be identical with that prevailing in other parts, except for an increased malignancy, due prabably to climatic conditions.

Up to this time the malady was generally known as "Redwater disease," a peculiarly unfortunate name, since it was based on a phenomenon which is only an incidental, and by no means invariable, symptom of the disease. But, far more important than this, the name "Redwater disease" conveyed no hint or warning of the nature of the impending plague. Could it have been from the first clearly recognised and distinctly described as identical with Texas fever, its danger would have been at once realised, and the first quarantine regulations proposed, or even more stringent ones might have been rigidly enforced, and the disease isolated or perhaps stamped out in the small area in which it first appeared in Queensland.

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[1] Extract from "Report on Tick Fever," by Dr J. Sidney Hunt and Mr William Collins: Brisbane, 1896.

Such knowledge, however, was not at that time available, and the purpose of this report is to present some outline of the investigations which have led to the positive identification of the Queensland disease—most aptly and appropriately called "tick fever"—with that known in America as "Texas or Southern cattle fever."

Little in the way of original observation had up to this time been published as to the essential nature of "Redwater disease" in Queensland; and though ticks had been stated to be the agents by which the disease was communicated, no experimental evidence had been adduced in support of this assertion, nor had the view found anything like general acceptance. The first object, therefore, was to endeavour to ascertain conclusively, by careful experiment, whether the disease was in reality due to tick-infection or not.

To this end a calf about six months old was, on the 15th September, infected with a large number of young ticks that had been raised (incubated) in the laboratory. The parents of these young ticks had been gathered from different animals of a mob of cattle amongst which the specific disease was very prevalent. They were hatched on the 11th September, and were consequently four days old when placed on the calf. By the 6th October they had become fully developed, in large numbers, upon the animal, and by the 10th had almost all fallen off. Before any large ticks were to be found, however—*i.e.*, during the last three days of September—the calf was found to have a certain degree of fever. It was drowsy and weak, and was observed to be constantly lying down. When compelled to move about it had a rather tottering gait, and swayed somewhat in its hindquarters. Its bowels were deranged—motions dark-coloured, and coated with small quantities of blood-stained mucus. Its temperature varied from 103·5° to 104·5° Fahr. Microscopic examination of its blood (in the fresh state) revealed the presence of peculiar amæboid bodies (to be presently described) in a certain proportion of the red blood corpuscles, and a larger number of similar bodies free in the serum. The fever lasted about twelve days. The calf became, for a time, thin and weak, but eventually regained its health and condition. It is worthy of note that, though six successive generations of ticks were subsequently raised on this calf, which was used as a kind of cultivating ground to keep the laboratory supplied with ticks, no ill effects whatever were manifest after the first infection. Its blood, however, when examined from time to time, showed always the presence of the extra-corpuscular motile bodies just now referred to.

This experiment would doubtless have been more striking had an aged animal been employed, because the disease runs a much more violent course in adult than in young cattle.[1] Subsequent experiments in artificial tick-infection, however, gave always the same results in respect to the production of fever and appearance of motile bodies in the blood. A large number of observations were also made, for purposes of comparison, on the blood of healthy tick-free cattle, but in none of these was there discovered anything resembling the motile bodies found in tick-infected animals.

The view that ticks were the cause of the so-called "Redwater disease" was therefore held to be justified.

The investigations at Hughenden were at first much hampered by the want of proper yards and appliances, without which adult bush cattle could not be manipulated; and also by the fact that few cases of the disease found their way so far as Hughenden. The first difficulty was overcome by the erection by the Government of excellent and substantial yards and other conveniences for the work of an experiment station. And the latter was speedily overcome by the increasing numbers of affected animals that arrived at the railway trucking yards from the infested districts. The experimental station paddock was so conveniently placed that any cattle found sick in the yards could be easily drafted into it. And in this way it was not difficult to obtain a sufficient supply of animals in all stages of the disease.

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[1] There does not, however, appear to be any evidence that these are less *susceptible*. Calves suffering from a first tick-infection have been observed to have a temperature of 106° to 107 Fahr. without showing signs of serious illness or distress. Their comparatively low mortality is universally recognised.

The disease is recognised by the following symptoms. In the first place, the animal may be seen to be, more or less, tick-infested. But it must be borne in mind that, as in the case of the artificially tick-infested calf just now referred to, fever is set up by young ticks before they have grown large enough to be discoverable to ordinary examination; so that it must not be supposed that tick fever is necessarily accompanied by *obvious* tick-infection. And in reference to this matter it may be here stated that, where one or two animals of a mob are seen to bear ticks, the remainder must always be regarded as also infested, though to prove it may require a more searching examination than is generally practicable on living animals. The first definite symptom of the disease, however, is increased body-temperature—fever. This may be present to a moderate degree for some days before any other symptom is apparent, though for obvious reasons it is not generally recognised except in the case of animals under close observation. After a while, generally from the second to the sixth day of the fever, the animal becomes restless, and may be seen to wander about as if anxious or uneasy. A little saliva runs from the mouth, though the nose is dry. The eye is bright and full, but soon loses its natural expression of bovine placidity, and acquires a rather wild and "haunted" look. The animal loses its appetite, though it still wanders about as if seeking food. It ceases to ruminate. In cows the secretion of milk is early suppressed. The urine begins to acquire a darker tinge, and is passed very frequently. The bowels are constipated, and the motions darker than normal.

As the disease progresses the animal ceases its restless wandering, and may be observed to lie down and get up again very frequently. It stands with its head down and its ears drooping. It seeks shade, and where a lagoon or other water is available will remain constantly standing in it. All the symptoms gradually increase in severity. The skin is now markedly hot to the touch. The coat is rough and staring. The breathing is hurried. The animal becomes drowsy, and is not easily disturbed, though when aroused it will sometimes charge fiercely. Though the eyes remain clear and open, the pupil will, from this stage onwards, be seen to be a good deal dilated; and the beast has a peculiar, dull, vacant expression, as though seeing, it saw not. In the typical acute disease the urine acquires a still deeper colour, passing from red to brown or almost black. It contains much granular *débris*, but does not generally give the reactions for bile pigment. It contains albumen in variable amount—there is frequently so much as to make the urine semi-solid on boiling. The characteristic micro-organisms have not with certainty been observed in it. The bowels remain constipated as a rule, with the passage of hard dark fæces, often coated with mucus, which is in many cases blood-stained. In some instances there is a violent spontaneous purging, and when this sets in pretty early in the attack it is by some regarded, with apparent justice, as a favourable indication for eventual recovery. The animal, from almost the commencement of the fever, looks hollow about the flanks, but the acute disease runs its course too rapidly to permit of much general wasting. In such as recover extreme wasting is a characteristic sequel. Muscular weakness, however, is an early and marked symptom, and the affected beast may be seen to sway in the hindquarters as it walks or stands, and more particularly as it turns.

In the last stage the animal generally becomes torpid, and apparently unmindful of all around it. It stands with its head lowered to the ground, breathing heavily, or leans for support against a convenient tree or fence. Its muscles, especially those of the flank and thigh, quiver and tremble from extreme muscular debility. If it be now forced to move, its swaying walk will be seen to have degenerated into an uncertain stagger. In some few cases the final fall is preceded by a stage of furious excitement. The duration of the disease is very variable. Some animals succumb as early as thirty-six to forty-eight hours after they are first noticed to be ill. But in animals that have been experimentally inoculated and carefully watched the fever has generally been found to commence from four to six days after the inoculation, and to last from six to eight days or even longer. A very sudden fall in the temperature precedes the fatal termination or commencement of convalescence, as the case may be.

Animals that survive a first acute attack are left extremely weak, and waste rapidly. In some cases, especially where they are free from constant fresh tick-infection, they gradually regain health and condition. In others a chronic fever succeeds the acute attack, and they steadily fall away and finally perish of exhaustion. The phenomenon of "redwater" is in such cases totally absent, and the mucous membranes, such as those of the lips, palate, and tongue, are pale and bloodless.

In another set of cases, animals that have survived a first acute attack, and have even to a great extent recovered from the subsequent debility, may suddenly develop a second acute attack, which is very frequently fatal. Such recurrent attacks have been sometimes regarded as relapses, but are more probably due to fresh infection by ticks.

Animals suffering from tick fever in any stage or form are apt to drop dead quite suddenly under circumstances of exertion or excitement.

The *post-mortem* appearances of the acute disease so exactly coincide with those of Texas fever, that any further description would be useless. The only points in this connection that seem to call for remark are that, while jaundice is described by the American authors as very rare, it has in greater or less degree been the rule rather than the exception in the cases observed in Queensland. Moreover, petechial hæmorrhages beneath the skin and serous membranes of the abdomen and chest have been more frequently noted than appears to have been the case in America.

The *post-mortem* appearances of cases that have succumbed to exhaustion following the acute attack, or to a second acute attack before they have completely recovered from the first, or to the combination of these conditions such as is sometime found in animals subjected to constant and excessive tick infection, differ from those of the typical acute disease in that there is less marked congestion of internal organs. The blood is thin and watery. The muscles are pale and bloodless. The kidneys and liver are light in colour. The urine is little or not at all discoloured. The bile, however, is frequently thick and dark, and the spleen may be considered enlarged.

The microscopical appearances recorded in the American observations refer very largely to the examination of sections of tissues, and more particularly to dried and stained cover-glass preparations. Those made at Hughenden—previous to acquaintance with the American work—were, for the most part, carried on with blood and tissues in the fresh state. In the former work attention was chiefly directed to the blood corpuscles and the stainable bodies found in them; in the latter to the free (extra-corpuscular) motile bodies. Both of these are doubtless forms or stages of one and the same organism; but the study of the free forms has resulted in the recognition of certain matters (to be presently detailed) which do not appear to have received attention at the hands of the American investigators.

Since motility is the most characteristic feature of the extra-corpuscular forms, it is obvious that examination in the fresh state is essential for their study. The usual staining methods (by which this feature is necessarily destroyed) have been found of comparatively little service.

For the examination of these forms in the blood of a living beast, it is sufficient to obtain a droplet, with the usual antiseptic precautions, from a pretty deep skin incision. It has been found more convenient to use a platinum loop to place the droplet on the cover-glass than to touch the exuding blood directly with the latter, as is generally done in examining fresh human blood. It has also been found of advantage in all fresh preparations to immediately seal the edges of the cover-glass by brushing round them with melted hard paraffin, as by this means evaporation and the currents due to it, which are often troublesome and misleading, are prevented.

Instead of mounting the blood directly, it has been found sometimes convenient to collect it in capillary glass tubes, the sealed ends of which are broken off immediately before use with a pair of sterilised forceps, and re-sealed in a flame as soon as the blood has run into them. In this way specimens can be collected easily and quickly, and examined at leisure, and under conditions more favourable than are generally obtainable in the neighbourhood of a stockyard. This method has, moreover, special advantages for the examination of the extra-corpuscular bodies: a droplet of blood mounted directly on to the slide must necessarily be very small in order that the corpuscles may lie quite flat and separate, and the minute size of the droplet reduces the chances of finding any of the bodies sought if, as is often the case, they are present in very small numbers. Moreover, where a very minute droplet is employed the cover is apt to be drawn by capillarity very tightly against the slide, and the compression so produced is sometimes sufficient to arrest the peculiar movements by which the extra-corpuscular bodies are, under other circumstances, at once recognised. By employing capillary tubes, both these difficulties are obviated. The blood, of course, clots in the tube, but many of the extra-corpuscular bodies are left free in the serum, which can be examined in any sized droplet that may be convenient. This method has also been available for the examination of the blood-cells, since a considerable number always escape the meshes of the clot, and a relatively large proportion of serum is no great disadvantage. It is perhaps worth mention that in tubes kept for some hours in warm weather the phenomenon of "phagocytosis" has been observed—*i.e.*, the leucocytes or "white blood-cells" have taken up or incorporated large numbers of the extra corpuscular bodies.

The number of free bodies to be found in the circulating blood is always limited. Three or four may sometimes be seen in a single field of the microscope, or a long and tedious search may be required before one is encountered. In the substance of the kidney and spleen, however, they are enormously abundant, and in a less degree in the liver. A satisfactory way of examining them in such situations has been to place a drop of sterilised (·5 per cent.) salt solution on the slide, and into this introduce a minute scraping, taken with the usual antiseptic precautions, from the substance of one of these organs that has just been removed from an animal killed in a late stage of the disease. When such a preparation is covered and sealed as before described, it will be seen, when placed under a magnifying power of about 750 diameters to swarm with the bodies in question.

These free bodies vary greatly in size, some being no more than $\frac{1}{30}$ and some as large as $\frac{1}{3}$, the size of a bovine red blood-cell, which is somewhere about $\frac{1}{4000}$ of an inch. They vary also in form, the majority being round or spherical, some pear-shaped, some oblong, some sausage-like, others constricted like an hour-class, and others irregular. None of these forms are by any means constant, since the bodies are perpetually changing their outline. They differ also in respect to colour; most are colourless and highly refractive, others have a yellowish or even reddish-brown tinge, but there is never any granular pigment. The majority appear homogeneous, others dark-centred, though this latter appearance may be due to their high refraction. A certain proportion are motionless, but the majority are in very active movement, and may sometimes be seen to work their way across the field of the microscope, apparently urged along by a flagellum. The most general and characteristic movement, however, is neither amœboid nor locomotive, but consists of a peculiar rolling on their own axes, which gives them a twinkling appearance, something like that of a small bright coin as it sinks in deep water.[1]

The intra-corpuscular forms of the micro-organism observed at Hughenden correspond exactly to those described in the American Report as the micro-organism of Texas fever.

In the circulating blood only a few corpuscles will be found invaded, and in these the micro-organism will be seen as a pale, well-defined, well-rounded body occupying about one-fifth of the corpuscle. Those observed in the cells of the general circulating blood have always been single. They undergo constant and very obvious amœboid movements or changes of shape, and also change their position in the body of the invaded corpuscle. Their movements, however, are very slow as compared with the movements of the extra corpuscular forms.

In blood from the capillaries of internal organs a large proportion of the corpuscles may be seen to contain pale bodies, varying in number from one to six. Most commonly there are two bodies in each cell, and these are more or less pear-shaped in outline, and are non-motile.

For the examination of all intra-corpuscular forms, staining methods are of great value. No satisfactory specimens, however, have so far been demonstrated in Queensland by the methods advocated in the American Report, possibly because the samples of methyl blue employed have not been of the proper quality. A good deal of work was accordingly required at Hughenden before a really satisfactory method of demonstrating the intra-corpuscular forms was discovered. The plan finally adopted, and one which gives really excellent results, was as follows:—The cover glasses on which the films are to be spread —each one in a separate watch-glass—are placed in any convenient oven and kept there till the moment the film is to be spread on them. This method of spreading blood-films *on hot cover glasses* has been found of very great advantage in all blood examinations. It ensures the *immediate* drying of the film, which is the all important point if the natural contour of the red cells is to be perfectly preserved. After the smears have been made, the covers are at once returned to their watch-glasses in the oven, and left there for a quarter of an hour or more. They are then ready for staining without any further heating. Before staining, however, it has been found advantageous to immerse the preparation for one or two minutes in absolute alcohol. From this it is taken and held for a few seconds till the alcohol has evaporated or been removed from the unprepared surface. It is then floated for five to ten minutes on a solution of gentian violet—twenty drops of a saturated alcoholic solution to half a watch glassful of distilled water. Heating the solution is undesirable, as it generally results in excessive over staining. The cover is next rinsed in water, and placed, for preliminary examination, under the microscope, to ascertain the exact degree to which the staining process has gone. Generally the corpuscles will be found to be of a uniform dark-violet colour, in which nothing whatever can be distinguished. The next step is to dip the preparation for a few seconds into very dilute alcohol, and again rinse and examine. The weak alcohol removes the dye from the substance of the corpuscle much more readily than from the contained micro-organisms, so that after a few dippings the colour of the corpuscle is reduced to a pale mauve, against which the micro-organisms, still holding the deep-violet dye, show up in strong relief. The preparation is then dried and permanently mounted in Canada balsam dissolved in xylol.

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[1] These appearances were reported to the chief of the Stock Department on the 10th October, 1895, and were shortly afterwards published in the *Queenslander*; and on the 29th February, 1896, were demonstrated to Mr C. J. Pound, the Government Bacteriologist, and substantially endorsed by that able microscopist, especially with reference to the presence of the flagellum.

The best preparations have been obtained from blood expressed from the capillaries of the heart's substance. They should be made the moment the heart is removed from the animal. Fairly good specimans have also been made from the capillaries of the kidney. Preparations from the capillaries of other organs have been less satisfactory.

Since tick fever is produced by infecting an animal with young ticks, and is accompanied by certain micro-organisms, in the blood, which are not found except as the result of tick-infection, the inference seemed irresistible that the micro-organisms or their ancestors must, in some shape or form, exist in or about the young ticks. It is hardly necessary to point out that the discovery of the micro-organism *in the body of a tick taken from a beast*—of which, however, there was no record—would be of little or no value in respect to the conveyance of the infection by young ticks since in such a case the inference would naturally be that any of the micro-organisms discovered had been extracted with the blood directly from the affected beast. It was therefore necessary to devote attention, in the first instance, to the examination of the newly hatched ticks. If the micro-organism was to be found in them, it must necessarily be in the free (extra-corpuscular) form. The free forms, however, as already pointed out, have little to distinguish them apart from their peculiar movements; and, since staining methods were found useless for their identification in fresh blood and tissues, these methods did not seem likely to be of much service for their recognition in the body of the tick, where the difficulties are greatly increased by the presence of all manner of granular matter, from which, in stained preparations, the bodies cannot be distinguished.

If, however, the method of examination described in the case of fresh tissues be adopted, and the larval tick placed on a slide in a droplet of saline solution and squashed under a cover glass, and the latter sealed with paraffin, certain bodies will be seen which, in their varying size and peculiar movements, correspond to those found in the blood and tissues of affected animals when similarly examined.

It may be said that such observation is insufficient for definite identification; more particularly in view of the fact that neither in America nor at Hughenden has it been found possible to produce tick fever by injecting beneath the skin a solution made by bruising up many thousands of ticks in the larval stage. The suggestion may, perhaps, be hazarded in passing, that the micro-organism requires to undergo certain developments in the body of the tick before it is in a condition to produce disease in an animal. Strong confirmatory evidence, however, of the identity of these bodies is to be found in the fact that they are present in certain parts of adult male ticks, in which situation their appearance is so peculiar and so characteristic as to leave little room for doubt on the matter.

The question naturally arises—What is the essential nature of these bodies, whether occurring in the bullock or the tick? The authors of the American Report have regarded them as belonging to the *Protozoa*, probably to the subclass *Sporozoa* or *Gregarindiæ*; and a very large proportion of the time and attention occupied in the investigations at Hughenden was devoted to the endeavour to ascertain the correctness or otherwise of this view, and more particularly the precise relation of these bodies to the tick. Since, however, the matter is not yet conclusively settled, and would involve a very lengthy discussion, it is not here entered upon.

Many attempts were made to cultivate these organisms, both from ticks and from the blood and internal organs of affected animals, upon the various solid and fluid substances generally employed in the study of bacteria, but without success. Most of the tubes remained sterile, or only developed such growths as were regarded as, and, in many instances, proved by experiment to be, only accidental contaminations.

The life-history of the Queensland cattle tick has been found to coincide with that of the tick described in the American work. Cattle ticks have been observed in many parts of Queensland to attach themselves to horses, and in some instances to mature on them; and at Hughenden sheep placed in a pen that had been previously occupied by tick-infested calves also became infested, especially about the face and under the arms, with ticks that came to maturity. No evidence has, however, been adduced to show that either of these animals suffer from the specific fever as a result of tick-infection.

This apparent immunity it was thought might be due to vigorous local resistance, which prevented the introduction of the infection into the system of these animals; for it was noticed that when they were attacked, at any rate for the first time, the point where each tick adhered was marked by considerable inflammation, as shown in the case of a sheep by an elevated pimple surrounded by a very red area, and in the case of a horse by a pretty extensive swelling of the skin and tissue beneath it. The well-known fact that in many diseases of microbic origin general susceptibility is inversely proportionate to local resistance seemed to lend colour to this idea.

With a view of ascertaining whether they were absolutely immune—when all possibility of protection by "local resistance" was removed by introducing the infection directly into the veins—or whether they were subject to the disease in a mild (attenuated) form that might possibly be useful for purposes of protective inoculation of cattle, the following experiment was performed:—Ten ccm. of blood from a bullock, very ill from tick fever, was injected into the jugular vein of a horse, and 6 ccm. into the jugular of each of two sheep, and, for purposes of control, 10 ccm. into the jugular of a healthy bullock. All these injections were made on the same occasion from the blood of the same animal. As a result the control bullock developed a very severe attack of acute tick fever. The horse remained unaffected. The sheep both developed high fever, the temperature in one case reaching 108° and the other 107° Fahr. (the normal temperature of healthy sheep being 100-103° Fahr.). One eventually recovered. The other was killed in a dying state on the tenth day after the injection. The internal organs, so far as it was possible to examine them at the time, presented none of the conditions found in cattle killed in the late stage of tick fever. Nor were any of the characteristic "double pear-shaped" micro-organisms found in any of its blood corpuscles. A certain number of these, however, were seen to contain very minute marginal bodies, similar to those of chronic tick fever. The blood of one of these sheep injected into a bullock produced no well-marked disease. This matter, in common with many others relating to tick fever, requires further study.

It may be mentioned that ticks taken from horses and sheep lay fertile eggs, but circumstances prevented the completion of the intended observation as to whether the young ticks hatched from these are capable of producing disease in susceptible animals.

An attempt was made to ascertain the length of time young ticks could survive on a pasture. The method employed was to place newly-hatched ticks on growing tussocks of grass under properly ventilated bell glasses. It was found that they did not live so long under these conditions as in empty glass jars. In the latter situation they were stated in the American Report to have been alive at the end of twenty-two weeks. But at Hughenden they have not survived more than half this time—perhaps because the heat of North Queensland is less favourable to them, under these conditions, than the cooler climate of Washington. Mr Pound has also observed that they live longer in jars in cool weather than in the heat of summer. This, as a matter of great practical importance, seems to be well worthy of further study, and might be worked out by any person who would keep exact notes.

Various substances were tested and reported on, as dressings to destroy ticks: none of these were absolutely satisfactory. The destruction of *all* ticks upon a beast is doubtless a great desideratum for certain purposes. But the discovery of a means by which tick-infection may be prevented for a considerable time is of at least equal importance.

In the absence of any satisfactory method of protecting cattle from ticks, attention was devoted to such methods as seemed likely to offer a means of protecting cattle from the disastrous effects of tick-infection.

The fact that animals reared in infested places, where they are constantly inoculated by ticks, do undoubtedly become immune, together with the fact that the disease can be artificially induced, seems to indicate very strongly that immunity may also be established by artificial inoculation. But even if inoculation by direct transference of blood should prove a protection against future attacks, the severe illness, loss of condition, and high mortality that have so far been the immediate results of this method are too serious to admit of its general application. It seems probable, however, that some less severe form of inoculation might be found which would be free from these drawbacks; more particularly some method that would more closely imitate the gradual immunisation brought about in nature by chronic tick-infection.

An attempt was made at Hughenden to ascertain if the disease could be attenuated by passing it through a series of healthy bullocks: the idea being that the disease might tend to "wear itself out" if passed on directly from beast to beast without being allowed to pass through the organism of the tick, which it was thought might be the means by which, under ordinary circumstances, its virulence is maintained or reinforced. The experiment was unfortunately not sufficiently extensive to give any very conclusive results. In a series of seven bullocks consecutively inoculated the one from the other, the first and sixth of the series succumbed to tick fever. To arrive at a definite conclusion in this matter it would probably be necessary to employ at least three or four animals for each step of the series, and so eliminate such sources of fallacy as arise from the varying conditions of health and natural susceptibility of individual animals.

The points of analogy between tick fever and the malarial fevers of man are so many and obvious as to at once arrest attention. Both are due to amœboid blood parasites which occur as intra and extra corpuscular forms; in both there is a long continuance of the micro-organism in the blood; both present themselves in acute or malignant and in chronic or mild forms; both are characterised by blood destruction and enlargement of the spleen; both are liable to recur; and lastly, both are associated with insects.

It is possible that the specific remedies for malarial infection, such as quinine, arsenic, carbolic acid, etc., may yet be found useful in the treatment of some cases of tick fever; but the doses would need to be large. Four grammes of quinine injected hypodermically into a tick-fevered calf at Hughenden produced no appreciable effect; and half a gramme of carbolic acid injected into each of two cows on the coast, whose temperatures were respectively 107° and 107·8° Fahr. at the time, did not avert a fatal issue. Obviously such experiments are of small value unless carried out systematically and on a considerable scale.

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PNEUMONIA AND PLEURISY IN THE HORSE.

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(*Continued from p. 47*).

Sequels of Pneumonia.—The only complication I have met with in pneumonia is rheumatism. In the few cases I have seen the affection was confined to the limbs, and shifted from one to the other. In one or two of the cases the convalescence was greatly protracted owing to the acuteness of the attack.

As the treatment of pneumonia is so closely connected with that of pleurisy, I propose to first consider this disease, and then to describe the treatment of pneumonia and pleurisy under one head.

PLEURISY.

Pleurisy may be, but very rarely is, an uncomplicated disease ; in a very few cases nothing but the pleural surface of the ribs or lungs may be affected, but by far the most common condition is a combination of pleurisy with pneumonia. We shall, however, endeavour to study pure pleurisy in the same way as we have studied uncomplicated pneumonia.

The pleural cavity belongs to the lymphatic system, and may in fact be regarded as a large lymphatic space. The cavity is divided into a right and left half, and the partition in the majority of animals is complete, viz., there is no communication between the right and left sacs. In the horse, however, this is not so, for we generally find one or more foramina in the mediastinum, which admit of a communication between the two sides of the chest. The opening in the horse is in the posterior mediastinum, about one-third of the way up. Once this opening is reached by a fluid in one sac, the material must be able to find its way into its fellow ; in this way a single-sided pleurisy might become double. I am bound to confess that in my experience such an extension of disease does not apparently occur ; in fact, pleurisies are from the first almost invariably double, and the foramina in the mediastinum are hardly likely to assist in extension, as they are blocked with lymph early in the attack.

Causes of Pleurisy.—The causes of pleurisy are nearly as obscure as those of pneumonia ; perhaps the theory of cold on a heated skin is better exhibited in the one than the other, but it is quite certain that there are many cases of pleurisy which it is impossible to trace to any definite cause. Of the definite causes next to exposure to cold comes that of the strangles virus, in fact, in young horses I am not sure whether the virus of strangles should not occupy the first position.

Symptoms.—An attack of pleurisy comes on suddenly ; the animal, perhaps previously in perfect health, is found shivering, dull, respirations rapid, pulse quick and very small, and a high body temperature. I think perhaps the onset of the disease is more rapid than in uncomplicated pneumonia, for I have in many cases been able to fix with almost absolute certainty the very short time, sometimes an hour or two, elapsing between perfect health and acute pleurisy. This is very different from pneumonia, in which, as we have shown, an incubative period is frequently present. The symptoms are more urgent than those of pneumonia, owing, perhaps, to the pain suffered. The breathing certainly is more distressed, and often from the onset laryngeal movements are present, which are only rarely seen in pneumonia.

The laryngeal movements referred to are an up and down, or to and fro motion of the larynx during respiration, and I am rather inclined to attribute this to the pain suffered.

Pneumonia is not a very painful disease, but pleurisy on the other hand is, especially the first few hours of the attack. Nothing is more common than for an attack of pleurisy to be ushered in with colicky pain, and such symptoms have thrown many a practitioner off his guard. The pain never lasts long and is rarely acute. These symptoms are rapidly followed by others which draw attention to the chest, foremost of which is an increase in the respirations.

Pleurisy, like pneumonia, commences below ; it is rare that the whole pleural surface from one end to the other is affected, but generally, at any rate early in the attack, the disease is confined to the lower and anterior parts of the chest, and the lower half of the diaphragm. Later on the disease may creep up the chest-wall and diaphragm, especially the latter, but fatal cases may often be found (one is thinking more of pleuro-pneumonia than pure pleurisy) where no extension to the top of the chest has occurred.

Changes in the Pleura.—Before dealing further with the symptoms of the disease, it may conduce to clearness if we study the naked-eye changes occurring in the pleural membrane as the result of inflammation, and the first of these is that the membrane loses its colourless, moist, and transparent appearance, it becomes dry and dull, and injected with minute vessels. Where all the vessels suddenly come from is a marvel, which can only be compared to the extraordinary rapidity with which the conjunctiva can become injected. On the costal surface the largest amount of injection occurs as a rule between the ribs and not on them, so that the intercostal space is often mapped out with great accuracy by injected vessels. It is probable that the acute stage of congestion is the most painful period of the disease, and that the pain and symptoms are relieved by the vessels exuding material through their walls.

All pleurisies do not terminate in exudation, and an extremely important point arises as to how long a dry pleurisy may last, or in other words, when may exudation occur? The answer to this question is that exudation may occur in thirty-six hours (and in one case I believed it to have occurred in twelve hours), but, as a rule, there is no indication of fluid existing in the chest until the second or third week of the attack.

It does not follow that no fluid is present in the chest until about the time named, but it is at this period that respiratory distress suggestive of hydrothorax is generally seen. It may be that the fluid is slowly accumulating, but I think my experience leads me to regard the effusion as sudden rather than gradual.

During the first days of a pleurisy the condition is one of intense engorgement of the vessels and loss of brilliancy in the membrane, which becomes dull and lustreless ; in the case of the pulmonary pleura the membrane becomes opaque and its surface faintly roughened. On the costal pleura membranes may appear, but whether these membranes are entirely produced by an exudation directly from the vessels, or formed as the result of the outpouring of fluid, is a question which will be considered presently. In the meantime we may note that if the membrane be peeled off the ribs, it discloses a surface which can only be described as resembling both in colour and texture a piece of maroon velvet.

Before discussing the character of the so-called false membranes of pleurisy, we must consider in what way they are produced, and it appears to me that we can account for their production in two ways, (*a*) The direct formation of a membrane on the surface, such as occurs in diphtheria. (*b*) The deposition of fibrin from the inflammatory fluid which is poured out. Both of these processes may occur in pleurisy. The one to come first into operation is *a*, viz., the direct formation of a membrane on the surface, but I believe that the bulk of the membranes found after death, especially these free in the chest, are caused by *b*, viz., by coagulation of the effused serum, and the production of fibrin. This fibrin forms a loose layer over both lungs and diaphragm, and is obviously unattached excepting at points to the underlying structure.

I think method *b* accounts for all the loose membranes found on the lungs and diaphragm, while all the permanent or attached membranes are probably produced by method *a*.

The evidence that membranes are formed from the fluid by the deposition of fibrin, is that the fluid drawn from the chest can rarely enter into spontaneous coagulation, even on the addition of fibrin ferment. This shows that all the fibrinogen has been converted into fibrin, while the fibrin is represented by the false membranes found within the chest, either partly attached or free.

The production of membranes which closely resemble in appearance a rough Turkish bath towel is one of the characteristic features of pleurisy, and governs the future progress of the case. Membranes are *nearly* always accompanied by an effusion, but effusion into the chest may exist without membranes. By means of these membranes the lungs are bound down to the diaphragm and to the chest-wall, not as the result of a general adhesion but only of a partial one, that is to say, the whole of the lung is not adherent to the rib-wall or diaphragm, but only portions of it. Further, we constantly find that what looks to be a large surface adherent to diaphragm or ribs turns out to be one adherent by its periphery only, while the centre is free and unattached, thus forming a sac in which serum may be contained.

The formation of these sacs is a very interesting feature from a clinical point of view, for in such cases relief of the exudation by puncturing the chest can only be effected to that sac which we have penetrated, while neighbouring sacs are unaffected by the tapping, and are full of fluid. In this way we can account for those cases which during life and after death give abundant evidence of effusion, and yet on tapping yielded very little fluid. This point will be referred to again.

Reverting once more to the symptoms of the disease, we observe that in some cases of pleurisy tremors or clonic spasms of the pectoral muscles, and sometimes of the triceps of the shoulder, are not infrequently observed. These may last for a day or even longer, and are quite unconnected with the muscular tremors or rigors which usher in the disease. The latter are over in an hour or two, but the former are very persistent.

The pulse is small and quick, often 60 to 80, possessing no character whatever, and not distinguishable from that of pneumonia. The respirations are quick, nearly always quicker than those of pneumonia, and they may be shallow or deep in character. Shallow breathing, that is, very little action of the abdominal muscles, is extremely suggestive of pleurisy, but there are many cases where this symptom is absent, so that it must not be relied upon as indicating freedom of the pleura from disease. Later on in the attack a ridge along the cartilaginous terminations of the false ribs may be very marked, especially if effusion has occurred, and in this latter condition the abdominal muscles if previously limited in movement are now active and very evident.

The temperature is high, higher as a rule I think than in pneumonia without pleurisy, and as in the latter disease the highest temperatures are generally registered during the first day or two.

Grunting elicited on percussing the chest or on turning the animal is often present, but as a sign of disease of the pleura it is most fallacious. An animal may grunt persistently throughout an attack of chest inflammation, and *post-mortem* examination reveal a perfectly normal pleura. I have verified this many times.

The cough in pleurisy is painful, low, weak, and suppressed. This character is due to the pain caused by a violent expiratory effort, so that the animal instinctively puts no force into the cough. I have met several cases where this characteristic cough was entirely absent.

Auscultatory Symptoms.—An examination of the chest by auscultation will, if the case be seen in time and the conditions are favourable for examination, at once reveal the nature of the attack. The so-called friction sound of pleurisy is present, varying from a gentle rubbing heard both during inspiration and expiration, to a harsh almost squeaking friction. The rubbing sound is heard through the phonendoscope with remarkable distinctness; if the palms of the two hands be rubbed slowly in opposite directions to and fro, a sound is produced not unlike pleural friction. At times the sound is so harsh as to deserve the term squeaking. This squeaking is quite different from, and could not be mistaken for, a pulmonary squeak, for the reason that it is continuous and of a different tone.

The continuous character of pleuritic rubbing is characteristic. I have previously drawn attention to it and attempted an explanation of the fact. It is again insisted upon here as there are many under the impression that a pleuritic rubbing is an inspiratory sound ; we have shown that it is both inspiratory and expiratory.

The frictional area will vary in extent; it is generally most marked behind the elbow, and in fact may be limited to this region, but as the disease extends the sound may be heard half way up the chest or even higher. Previously we have alluded to the remarkable fact that the friction in pleurisy may be most marked just behind the heart, and that the sound is synchronous with the heart-beats. This is suggestive of pericardial complications which are exceedingly common, but apart from this the heart-beat may act mechanically on the lung producing the sound, and this is supported by the fact that friction sound over and in front of the heart may be entirely lost, which we should hardly expect in pericarditis.

In uncomplicated pleurisy the respiratory murmur can be distinctly heard through the friction, but this is soon lost if the lung becomes affected or if effusion occurs.

The pleuritic rubbing only lasts a short time; it often disappears in a few hours, commonly after the first day, occasionally as late as the second. This is probably the explanation why it is so seldom heard, the practitioner not being called in to his case early enough.

It is important, however, to note that acute pleurisy may be present and yet undiscoverable by any auscultatory signs ; that is to say not a trace of pleuritic rubbing or friction may be present. I have observed this in some of my fatal cases, and it leaves us in the unfortunate position of having to acknowledge that an acute pleurisy may be present undetected by auscultation. I take it that the pleuritic rubbing in these cases is lost by early exudation.

Percussing the chest I find of very little use in the early stage ; later on it is valuable should effusion occur.

The subsequent progress of a pleuritic case is simple; the disease may terminate life in forty-eight hours, but as a rule it runs into the second and frequently into the third week before causing death. On the other hand, should resolution occur, we may look for it towards the end of the first or beginning of the second week. To follow the latter case is unprofitable; the pulse, respirations, and temperature gradually fall, the appetite improves, the animal is brighter and takes notice of what is going on, and the chest sounds indicate a return to the normal. From a clinical point of view the chief interest is centred in the case which is doing badly, or at a standstill, and in such we look for effusion; if that can be avoided they may recover, if it occurs they are almost sure to die.

The Signs of Effusion.— We have previously mentioned that effusion may occur as early as twelve hours, but such is rare; slow effusion may be going on for some days, the existence of which the practitioner may suspect from a certain degree of dulness of the chest on percussion, and absence of sound on auscultation. But both of these features will be present in lung consolidation, and as by far the majority of cases of pleurisy are combined with pneumonia, it is not possible to say early in the case how far the dulness of the chest may be due to solid lung, and how much to effusion. If it is considered important to settle the point a needle may be introduced into the chest, but at the same time we must bear in mind that should the result of this be negative it does not follow that the chest is free from effusion, though the evidence is very strongly in favour of such an opinion.

Another symptom which points to effusion is a fall in the temperature and pulse rate, with an improvement in the general appearance of the case, but no reduction in the rate of respirations. Such symptoms, however, are very common in lung abscess, with an entire absence of effusion. An œdematous swelling of the brisket and inferior abdominal line is very suggestive of effusion, but the chief symptom pointing to effusion is the character of the breathing. The mechanical pressure on the diaphragm of a quantity of fluid necessitates a more vigorous action of the abdominal muscles, so that hurried and deep respirations are extremely suggestive of some quantity of fluid in the chest. I qualify the statement by using the term "some quantity," for it is certain that a relatively small amount of fluid may exist in the chest without any marked deepening of the respirations.

When, however, the quantity of fluid is considerable there is marked respiratory distress, distension of the nostrils, a peculiar movement of the abdominal muscles, turning out of the elbows to bring additional respiratory muscles into play, movements of the muscles on the front of the femur, just behind the flank, due to the energy of the abdominal muscles, and protrusion and retreat of the anus during inspiration and expiration. Of all these the peculiar character of the abdominal breathing is the most characteristic and absolutely diagnostic, so distinctive indeed that when present effusion can be diagnosed without even entering the sick-box. We must, therefore, study these movements, which are exceedingly difficult to describe, and in the first instance it will be well to note the rhythmical movement of the abdominal muscles in a state of health.

If the flank be watched during an inspiratory effort, it will be observed to descend in a steady regular manner, and during expiration to ascend slowly and regularly; the respiratory movement is a perfectly regular up-and-down motion, both the up and the down movement being absolutely true, viz., regular and unbroken.

It is not uncommon in disease to have the expiratory effort double, due to the fact that it is stopped half way and then sent on again. Such a condition resembles the double expiratory effort of emphysema, but, unlike it, disappears as convalescence becomes established. There are no special cases in which such double expiration are common; I have observed them both in chest and abdominal affections, perhaps more frequently in the latter.

As a rule, both in pneumonia and pleurisy, there is nothing specially characteristic in the respirations; they may be full or shallow, in pleurisy generally the latter, but they are nearly always true in their up-and-down movement.

When effusion occurs in pleurisy quite another state of affairs exist, and one most difficult to describe. I have attempted to describe it elsewhere, but am still unable to settle the doubtful points raised in that paper.[1]

The peculiar character of the breathing in effusion into the chest has long been recognised, and by many it is described as resembling that of emphysema of the lungs. This, however, is not strictly true; in each case there is a double upward movement of the abdominal wall, but there the resemblance ends, for both upward movements of the abdominal wall in emphysema are *expiratory* efforts, while in hydrothorax, one *upward* movement is *inspiratory* and the other expiratory. In hydrothorax we have a peculiar see-saw action of the abdominal wall, but no double expiratory effort.

During normal inspiration the diaphragm, principally at its upper half, recedes and carries back with it the abdominal viscera behind. The stomach, liver, and bowels are always advancing and receding during each inspiration and expiration.

In pleuritic effusion there would be no difficulty in the diaphragm receding if it were not for the adhesions contracted between it and the lungs. The fluid pressing against its anterior surface would give the retreat of the diaphragm mechanical assistance, but the adhesion of lungs to diaphragm, and the painful state of that muscle, limit its movements considerably. There is then a difficulty in inspiration, greatly exaggerated by the hydrostatic pressure to which the lungs are exposed.

The first movement of the abdominal wall in hydrothorax *inspiration* is not a descent of the whole wall as observed in normal inspiration, but the *ascent* of one part of the wall, and a descent of its neighbouring part. This is best understood by dividing the abdominal wall into two parts, a flank region in front of the femur and a sternal portion behind the sternum. Under normal circumstances these two regions move together and in the same direction, but in hydrothorax they move separately and in opposite directions; thus while one region is ascending the other is descending. In my original communication on this subject, I left it uncertain whether the inspiratory upward movement of the abdominal wall occurred at the sternal or flank region: there appears to be no fixed rule, for sometimes the inspiration is produced by an ascent of the sternal, at others by an ascent of the flank region. But the peculiarity of the breathing and the impossibility of making accurate observations on the subject without a trained observer as assistant, suggest that this difference may be due to error of observation, for by simply watching the abdominal wall it is impossible to believe that it is an inspiration which is occurring, and one can only be kept right in the matter by having an assistant at the nostrils who indicates at each moment which is the inspiratory, and which the expiratory effort.

Whether the flank or sternal region of the abdominal wall goes up first in the inspiratory effort need no longer be discussed; whichever it is, it is accompanied by a downward movement of the abdominal wall in the opposite region. Thus if the flank ascends and is drawn in during inspiration, at the same moment the sternal region descends; in this way is produced a see-saw movement which in my experience is absolutely characteristic of the existence of hydrothorax. During expiration the sternal portion of the abdominal wall ascends, and a well-marked ridge appears along the borders of the cartilages of the false ribs, while the flank portion descends and is pushed outwards.

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[1] "The Nature of the Respiratory Movement in Hydrothorax." Veterinary Record, Vol. iii., No. 332, 1894.

Briefly then, at *inspiration* the flank is drawn in by an upward contraction of the abdominal muscles, while the sternal region of the abdominal wall descends; during *expiration* the sternal region is drawn upwards, while the flank descends and is pressed outwards.

The in-and-out movement of the flank corresponding with inspiration and expiration is as a rule very marked, and extends back so far towards the pelvis that it places in motion the muscles above the patella and causes these to appear to move in and out, a phenomenon I have never observed excepting in hydrothorax.

Turning now to the general question of hydrothorax and the value of the respirations as an aid to diagnosis, it is to be particularly noted that fluid may exist in the chest without giving rise to the peculiar breathing we have been describing; in other words, it is not every case of hydrothorax which exhibits diagnostic abdominal breathing, so that the absence of this characteristic respiration does not afford undoubted evidence of the absence of fluid from the pleura. This is an extremely important point, and shows how necessary it is that the practitioner should not depend upon one single symptom. It is quite surprising the amount of fluid which may be contained in a chest without the see-saw movement being developed; in fact it would appear that the latter only comes into play when the chest has held all it can hold, for obviously there is some limit to the amount of fluid the chest will contain, since the lungs must have a certain amount of space allotted them.

It is clear that the intense compression to which the lungs are exposed in hydrothorax is the explanation of the difficulty in getting them filled with air, and that the weight of fluid pressing against the diaphragm is the explanation of the difficulty in expiration.

CHARACTER OF THE PLEURITIC EFFUSION.

The character of the pleuritic effusion depends upon the stage of the case at which it is examined and any complication which may exist. For example, fluid effused with extreme rapidity, say within the first day or two of attack, is often blood coloured; it would appear that the damage to the vessel wall is so great that not only plasma but also red corpuscles pass through. Under ordinary circumstances, where effusion occurs during the second or third week, the process is a much slower one, and nothing but perfectly transparent yellow liquid is obtained. Or again, if the pleurisy is complicated with necrotic pneumonia the fluid may be partly purulent, of faint or intensely fœtid odour, and extremely turbid. The yellow colour of the effused liquid is due to a fatty colouring matter termed serum lutein, and this frequently gives a greenish colour to the effusion by reflected, and a yellow by transmitted, light.

As a rule the fluid is non-coagulable, either on standing, whipping, or the addition of fibrin ferment; but I have seen some exceptions to this, and I regard an effusion which jellifies on withdrawal as one of recent production. The effusion which does not clot, on the other hand, is one which has deposited its fibrin within the chest, thus assisting, as previously explained, in the production of false membranes. A pleuritic effusion after standing twelve hours will often deposit a jelly-like clot, which lies at the bottom of the vessel.

I am not prepared to discuss why the walls of the pleural vessels leak under the influence of inflammation, but I do know that a leakage is not an absolutely necessary accompaniment of an attack.

PERICARDITIS.

All the time we are discussing the features of an inflamed pleura, we are thinking of the membrane lining the chest-wall and reflected over the lung; but this by no means completely represents the changes which occur, for it is impossible to make a *post-mortem* examination of a pleuritic case without finding the pericardium deeply involved, though only the external layer, viz., that formed of the reflected pleural membrane.

The pericardial sac is swollen, its layers thickened and œdematous, its surface deeply blood-stained, at times almost gangrenous, it is covered with membranes which give it a rough appearance, and becomes so altered by disease as to almost defy recognition or description.

This inflammation of the pericardium occurs quite early; it is in my experiences never absent, and it may account for the generally higher death-rate which pleurisy possesses. The internal surface of the sac is never affected with inflammation, but there is frequently fluid in it. The existence of a friction sound which may be pericardial has previously been mentioned.

In addition to a layer of lymph over the pericardium, lungs, chest-wall, and diaphragm, the same is found covering the vessels leaving and entering the heart, and the whole length of the thoracic œsophagus. I have often thought how painful this must render swallowing.

THE LUNGS COLLAPSE FROM HYDROSTATIC PRESSURE.

From the whole of the inflamed surface fluid may be exuded; this naturally collects first at the base of the chest in front of the diaphragm, as being the lowest point in the cavity, and from here it gradually rises. As it does so the lungs are exposed to pressure, as a result of which they collapse at the point where the pressure is greatest, and they lose by shrinkage the position they should normally occupy. In this way one-third to one-half the lung disappears, so that on opening the chest the only portion of lung found is that occupying the upper half or two-thirds of the chest. The anterior and inferior lobes on both sides are collapsed, and expose the pericardial sac, which from its thickening looks as if it occupied the whole of the anterior portion of the chest cavity.

The lungs are not only pressed up out of their place but they are floated up, for I have known a collapsed anterior lobe to be turned back and fixed by fibrinous adhesions to the base of the heart and the upper third of the lung, and it could only get into this position by floating in the fluid.

The effect of compression on the lung structure is that it becomes condensed, and presents the appearance of a piece of flesh; it is carnified and no air whatever exists in it.

The fluid in the chest does not present an absolutely level surface such as it would occupy in a vessel; it is higher at the sides, and here it comes up to a thin edge, the result of the movement of the lungs above, which are always attempting to compress it in a downward direction. Inspiration and expiration produce a constant ebb and flow in the fluid; the tide rises with *expiration* and falls with *inspiration.* This is exactly the contrary to what might be expected; but as the total capacity of the chest enlarges during inspiration, while the total amount of fluid remains the same, there being a greater space for its accommodation the tidal level falls, to rise at the next expiration. This may easily be proved by observing the pulsations of the fluid passing through the canula when the chest has been tapped; the flow increases and is discharged with much greater force during expiration, while during inspiration the jet becomes much smaller and the flow slackens. It is at the moment of inspiration that air is liable to enter the chest when the level of the fluid in the cavity reaches the level of the canula.

It is only in a few cases that the total amount of fluid effused is free to move about to any part of the chest; as a rule fibrinous septa, forming bags or spaces between lung and chest-wall and lung and diaphragm, are formed, and the level of the fluid in these sacs is of course unaffected by the general level of the fluid in the chest. In this way is explained the undoubted fact that a quantity of fluid is often found, *post-mortem*, exceeding that drawn off by operation shortly before death, although we may have been under the impression that the pleural sac had been nearly emptied by the operation.

THE RESPIRATORY SOUNDS IN HYDROTHORAX.

What is the condition of the respiratory sounds in hydrothorax?

This question brings us to the consideration of a curious point of great practical importance; in the abstract we might consider that the presence of fluid in the chest means a total loss of respiratory murmur of any kind, and up to a certain stage of the disease this is true, but as the effusion advances and the case gets worse respiratory sounds reappear even to the bottom of the chest. It is true they are not normal sounds but are of a markedly tubular character, but they are there, and experienced or inexperienced one is very liable to be thrown off their guard, and declare that the existence of any respiratory murmur, normal or abnormal, is not compatible with fluid in the chest. Such, however, is a complete error. *The chest may be half full of water, and yet the most perfect respiratory sounds may be heard behind each elbow*, though not a particle of lung is nearer than 5 or 6 inches of the spot in question.

This is the great practical lesson I have to present; it may be known to others, but I have only learned it during the last five years. The explanation is that fluid is a very good conductor of sound, and the respiratory murmur of the lung above is transmitted through the fluid below and heard at the bottom of the chest. The reason why the sound is only heard towards the end of the attack, appears to be that a certain condensation of lung structure is required, so as to bring the larger bronchi close to the level of the fluid, for the sound heard is undoubtedly tubular and not vesicular.

THE DIAGNOSIS OF EFFUSION.

The diagnosis of effusion depends upon the peculiarity in breathing, the intense wooden dulness on percussion, and the invariable presence of an in-and-out movement in the muscles clothing the front of the femur. The existence of tubular breathing at the bottom of the chest increases instead of decreases the probability of fluid being present.

All this time no mention has been made of the general symptoms of the disease. These are variable; the pulse may be small or fairly full, the appetite good or bad, the case may occasionally lie down or stand persistently, grunting may be present or absent, and the cough though nearly always short, painful, and suppressed, may occasionally be possessed of remarkable strength.

In very rapid cases there is considerable distress before the end arrives. The patient may lie down repeatedly for a short time, resting on the breast, rarely lying on the chest. After rising he may walk round, and even occasionally pawing with one fore leg is not an uncommon symptom of the intense difficulty in breathing which exists. The pulse is so small that it cannot be taken, the heart sounds extremely rapid, and the interval between them so short, that it is like the ticking of a clock. This latter condition of the heart may be apparent from the first moment of the attack, and as the case progresses the heart sounds appear to become more and more distant, probably owing to swelling of the pericardium.

A case may have pleurisy without any effusion. I have seen this repeatedly in equine pleuro-pneumonia, but I cannot conceive that a case of pure pleurisy would die from the disease unless effusion occurred. The effusion produces a great drain on the system, for the animal virtually bleeds into its interior, and further, by reducing the respiratory area and eventually compressing what is left, produces death by suffocation.

The treatment of pneumonia and that of pleurisy differ in no essential particulars, and we may well consider the two together, but the treatment of hydrothorax is a special matter and can most conveniently be considered here. In the first instance, what is the value of tapping the chest?

TAPPING THE CHEST.

The only argument in favour of tapping is the relief of dyspnœa; indiscriminate tapping is bad practice. It encourages what we have termed bleeding into the interior, for the fluid is replaced very shortly after being drawn off; in this way frequent tapping may hurry the fatal termination. The chief indication for tapping is respiratory distress, and this should be relieved by moderate tapping, so as not to unduly reduce the pressure on the thoracic vessels, and thereby favour effusion through their walls.

By far the majority of horses which require tapping die, yet inasmuch as one meets occasionally with recoveries after the operation the procedure is perhaps not without value. The main aim of our treatment should be directed towards preventing effusion, and this matter will be dealt with elsewhere. Once effusion has occurred our aim should be to favour absorption, and should that fail and life be threatened by hydrostatic pressure, we should puncture the chest and favour the gradual, not sudden, expansion of the collapsed lung.

Some years ago I made many observations on the treatment of hydrothorax. I first tried emptying the thoracic cavity as completely as possible of fluid, for this purpose puncturing the chest very low down. In addition to this transfusion was tried, the material from the chest being put back into the general circulation through the jugular vein, but it was of no use, and the return of the chest serum into the general blood stream was not without risk, for I once obtained intra-vascular clotting. After this accident I persisted in the transfusion, using a normal saline solution, viz. ·6 per cent. With it I had no success, though it certainly appeared rather a hopeful method of treatment. I have tried limiting the fluid consumed, but without success. It neither prevents exudation nor favours absorption.

We may say, therefore, that at present we have no treatment for hydrothorax. In an extremely small percentage of cases the progress of the effusion ceases, and in some even absorption of the effused material may occur, but such are extremely rare.

Absorption can of course occur from the normal pleural surface. Starling and Tubby have shown that the blood-vessels of the pleura absorb more rapidly than the lymphatics.[1] But the absorption of a fluid from the pleural sac which has the same composition as the circulating blood is a slow process; normal blood-serum, for example, is absorbed with extreme slowness.

These experiments receive clinical support in those cases where absorption of pathological fluid from the chest does occur; the process is a very slow one.

The operation of tapping is very simple and is so generally known and described that it need not be mentioned here. I endeavour to use the same puncture each time, no irritation is set up in the wound, and it saves trouble and a little pain to the patient, for the recently glued edges are easily forced apart by the point of the instrument, and a fresh cut in the skin is avoided. I draw off about six quarts of serum at each operation, and tap every day if the symptoms need it, using each side of the chest alternately.

The diet for a hydrothorax patient cannot be too liberal or too nourishing. I frequently give them their own blood-serum, this will be mentioned again in speaking of the general treatment of pneumonia and pleurisy.

POST-MORTEM APPEARANCES.

When, as unfortunately often happens, death occurs from pleurisy the *post-mortem* appearances are very regular and distinctive. On opening the chest gallons of fluid may escape, clear and free from odour, or turbid and fœtid. On removing the chest-wall, which may or may not be partly attached to the upper third of the lung, the whole or part of the pleural surface of ribs and lung are found plastered over with lymph, resembling in appearance a bath towel, much of which is semi-detached. If only the lower two-thirds of the chest-wall are affected the pulmonary pleural lesions will also be limited to this extent, but as a rule the whole of the diaphragm from top to bottom and from side to side will be found affected by the inflammatory process.

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[1] Absorption from and Secretion into the Serous Cavities: Journal of Physiology, Vol. xvi., p. 140.

The lymph or false membranes may be yellowish-white or grey, depending upon whether the fluid in the chest be aseptic or putrid; if the latter, the membranes are of a greyish colour. They are shreddy and fibrinous-looking, and on the lung in an old-standing case may closely resemble large warts in appearance. Quantities of false membrane may be found lying free in the cavity of the chest, or forming a sheet-like layer over the lungs or diaphragm which can readily be lifted up.

Owing to the peculiar character of the fibrinous membranes, cavities or sacs are frequently formed between the lung and chest-wall, and the lung and diaphragm. There may be several of these, each distinctly separated from the other, and with no communication of their contents. We have already alluded to these and their practical bearing.

Between the lungs and the diaphragm, which are more or less closely adherent, a considerable quantity of jelly-like serum can be found, and the same jelly-like exudate may be observed on the immensely thickened mediastina of the chest.

The lungs themselves are shrunken upwards to an extent which can only be realised by opening the chest from the side and removing the ribs. The shrunken condition exposes to full view the pericardium and vessels of the heart and the inferior mediastinum; in fact the contents of the chest appear to be represented mainly by heart, pericardium, and mediastinum.

The cause of this collapsed condition of the lung has been sufficiently dealt with above. The lung itself (assuming it to be free from pneumonia) is quite healthy, excepting where it is condensed by pressure; here on section the part is darker in colour than normal, and cuts and looks like a piece of flesh. Between the lung and the pleura is found, especially towards the inferior margin of the organ, a marked amount of yellow jelly-like exudate. In by far the great majority of cases the lungs are affected, in which case they present nothing distinctive from the *post-mortem* lesions which have previously been described. When the fluid in the chest is turbid and fœtid we may be sure that it is infected from some necrotic area in the lung, and that the pleura is probably perforated.

The pericardium presents some remarkable features. In cases which have died very early in the attack the sac externally is a highly thickened blood-stained mass which defies all attempt at description.

In cases of some little standing the same fibrinous membranes cover the pericardium as are found elsewhere in the chest, but between the several layers of the heart sac is an immense amount of serous exudate, and it is this which gives rise to the thickening of the pericardium.

No matter how extensive the damage may be to the outside of the pericardium, no injury will be found to its interior, though it may contain serous or blood-stained exudate. Only once have I seen the serous covering of the heart muscle itself faintly rough.

Such are the chief lesions found in an ordinary attack of acute pleurisy, and with this extensive disorganisation of a thin delicate serous surface it is no wonder that cases of pleurisy are so fatal.

PLEURO-PNEUMONIA.

According to the plan laid down at the beginning of this communication, a consideration of pleuro-pneumonia should follow that of pleurisy, but as a matter of fact such is not required. Though infinitely more common than pleurisy, there is no necessity for specially dealing with it, as we have considered in some detail the two diseases of which it is composed. Though our consideration of pleuro-pneumonia is limited to a few lines, yet we must bear in mind that in practice it is the most common condition in which we meet pleurisy, and further, it is the most fatal. The recoveries from uncomplicated pneumonia are numerous, from pure pleurisy, so far as we can judge, fairly frequent, but add a pleurisy to a pneumonia and the most fatal chest disease of the horse is at once exhibited. This will be very apparent from my statistical tables.

THE TREATMENT OF PNEUMONIA AND PLEURISY.

The confidence with which certain lines of treatment have been prescribed in pneumonia and pleurisy is only equalled by the positive assurance that opposite methods of treatment are certain to lead to favourable results. In spite of these assurances inflammation of the lungs and pleura is at present unchecked by medicine, and a serious source of mortality. Blood-letting, opium, calomel, aconite, antimony, stimulants, and counter-irritation, have each in turn or collectively been described as the sheet anchor in the treatment of pneumonia.

It must of course be obvious that no specific action can be claimed for any or all these methods of treatment; they can neither cut a pneumonia or pleurisy short, mitigate its intensity, nor stay its onset, and one or the other of these actions we might reasonably expect to follow the administration of a specific.

THE SEDATIVE AND STIMULANT SCHOOL.

The two schools in the treatment of pneumonia and pleuro-pneumonia may be described as the sedative and stimulant, and these are further complicated by the employment or non-employment of counter-irritation. I do not know that either of the opposite camps can give any sound reason for the line of treatment they pursue. The advocate of sedatives would probably point to their value in steadying the heart, reducing the frequency and increasing the force of each beat, while the stimulant school would probably point to the obstruction existing in the circulation of the lung, the tendency to heart failure, and the value of stimulants in preventing it. Again, the advocate of counter-irritation points to the relief this affords, without in any way explaining its action, while the opposite school maintain that it produces unnecessary suffering, and destroys the little chance of recovery left to a patient with pneumonia or pleuro-pneumonia.

In judging of the causes which lead two men, say of equal repute, to adopt different and opposite lines of treatment, we must bear in mind that no man takes up one method of treatment in order to be as hostile to his neighbour as possible; but he adopts it either as the result of previous bias in his early training, or because he finds from experience that when adopting this method of treatment he enjoys a larger share of success. Observe that I am careful not to say the success is due to the treatment, but that when a certain line of treatment is adopted success is greater. To many it may appear that if success is obtained it must be due to the treatment, but this by no means follows, excepting we be dealing with a disease which by every other method of treatment is attended by considerable mortality. Now, pneumonia by any and every conceivable method of treatment will be followed by a certain percentage of recoveries, and to prove that one method of treatment is superior to another we must be able to show beyond doubt that (1) a larger percentage of recoveries is invariably obtained, (2) that all the cases under opposite methods of treatment are of equal severity, (3) that the patients in both groups are of similar age, and are living under or have lived under equally favourable or unfavourable hygienic conditions, and (4), the personal equation of the observers must be abolished. When such a rigid test is applied to the treatment of a disease by different methods, it comes under the heading of a carefully arranged experiment, and the conclusions to be drawn from it are correct if the number of observations be sufficiently numerous.

We all know how very nearly impossible it would be, excepting in a large institution, to apply such rigid methods of enquiry, and if figures were available they would probably show that no matter what line of ordinary treatment be adopted the mortality in each group is much the same. I use the term ordinary treatment, to indicate that which is not employed with the express and known object of neutralising the poison on which pneumonia presumably depends.

So long as the treatment of pneumonia remains unlike the treatment of tetanus and diphtheria we cannot find fault with or blame our neighbour for whatever treatment he adopts; the practice of medicine is unfortunately in the main a purely empirical art, and everyone acts according either to his experience or his previous training.

In spite of the opinions we have expressed, it is permissible to review in some little detail those features in pneumonia to which treatment has been more especially directed, viz., the engorged state of the lung, the fever, heart failure, and nervous prostration.

TREATMENT OF THE ENGORGED LUNG.

Bleeding.—The engorged condition of the lung is always present, and it increases as the disease extends, but under no circumstance involves more than a portion of one or both lungs. The accepted view of the evil of engorged lung is the mechanical effect on the right side of the heart, leading to distension of the right ventricle, and so affecting indirectly the whole venous system. Further, it is supposed that as less blood is finding its way through the lungs, less reaches the left auricle, so that the body is starved of blood in its arterial system, and gorged in its venous. It was probably to remedy this state of affairs that blood letting was at one time so universally adopted. Under its action the character of the pulse was said to improve, and the blood pressure was lowered. We know, of course, that at the present day this is either not believed in, or only considered to be partly true; still it is a matter for consideration how far we are neglecting the experience of our ancestors, in entirely withholding moderate bleeding in well selected and early cases. The practice of moderate venesection in affording relief to the right heart is also I believe gaining ground in human practice.

At the same time I should clearly state that I cannot speak of the practice from my own experience, and further, I believe the heart distension to be considerably magnified, for so long as there is any moderately good available area of sound lung (and such we have shown to be nearly always present), the obstruction to the right heart must in time be relieved by an enlargement of the vessels of the sound portion of lung. Such a compensation in the venous system of other parts of the body is of common occurrence, and there is no reason why it should not take place in the lung. Further, even the most ardent advocate of bleeding in days gone by hesitated to put this practice into operation at a late stage of pneumonia, and yet, inasmuch as the lung obliteration is progressive, nothing can be more certain, if there is any truth in the obstruction theory, than that the right heart is more congested towards the end of an attack than at the beginning. I shall hope to show presently that there is very little reason for believing in the obstruction theory, and hence blood-letting cannot be recommended on this ground alone, nor can it be urged as affording relief by lowering blood pressure, for no fact in physiology is better ascertained than the influence of bleeding on blood pressure. The latter does not fall until two-fifths of the blood in the body have been abstracted, an amount which even the most ardent supporter of venesection would scarcely care to recommend, and the explanation is that the arterial system is self-adjusting, and regulates its capacity according to the total amount of blood in circulation, so that the blood pressure is maintained at a mean level. Finally, in connection with the question of bleeding, it must be remembered that the bulk of fluid in the circulatory system is very rapidly renewed, so that even were it permissible to bleed to the point of lowering pressure, the bulk of the blood would soon be raised to its original amount.

Of course the value of blood-letting may not in any way depend upon the reduction in blood pressure or relief to the lung or heart, but may have some other action unknown to us; it certainly seems strange that for so many centuries it should have held the foremost place in the medical mind; and reverting to a period well within the memory of thousands still living, it is quite certain that though the old practitioner was denied the knowledge of antitoxins and the like, he was nevertheless an acute observer of symptoms, and it is hardly conceivable that a practice which we regard as pernicious and harmful should have been performed by him unless some benefits were occasionally obtained. Have we too far pursued a course in the opposite direction? I am just as opposed to bleeding as the majority of human and veterinary practitioners, but the position is not a logical one, for it means I am opposed to a method of treatment I have never tried, and it only shows how difficult a thing it is to eradicate the bias of one's early teaching.

My point, however, respecting the congested lung is very simple, viz., that the congestion does exist, and that the obstruction in the lung increases with the extension of the disease, but that this does not to any serious degree overload the right heart, as compensation occurs. The congestion of the lung exists as an effect, and will disappear as the lung trouble clears up, but it does not occur to me that the overloaded lung can be treated through attacking the capacity of the circulatory system.

Counter-irritation.—Can the congested condition of lung be attacked by counter-irritation? This question opens a field of discussion which one instinctively avoids. Common as is the practice of counter-irritation, I think it permissible to say that no one can explain its action; it is one of the many empiricisms of medicine which, I believe, are absolutely unsupported by any experimental enquiry. Perhaps I may be permitted to speak in this tone with the greater freedom inasmuch as I have hitherto always employed counter-irritation in lung trouble; but when men with equal experience, equal powers of observation, and equal solicitude for the welfare of their patients tell us that they never employ the practice, it is calculated to cause us to pause, and ask ourselves the question whether counter-irritation is necessary or beneficial.

I cannot, however, go into this question; it is impossible to criticise a practice one has never regularly adopted, and it only shows how the same good can be reached by two opposite routes, how the same end can be attained by opposite measures, or, to put it more clearly, it shows how much interference the system will tolerate without seriously resenting it, and how extremely ignorant we are of the action of the most simple and common-place remedies.

Years ago one was taught to employ severe methods of counter-irritation, such as mustard mixed with vinegar or turpentine, and rubbed in until the operator was tired. I unhesitatingly condemn this practice as unnecessary, I cannot say as harmful, for I have no data to guide me. Through a long series of years I have gradually reduced the strength of these external applications to the chest, until I now use a mustard application, the virulence of which is greatly attenuated by mixing it with from three to six parts of flour, according to the character of the skin and the surface to be covered. In course of time I may reach that point when only flour and no mustard will constitute the application; it only shows how slowly one moves in changing his established practice, and it is probable that even this slow evolution of opinion is not without its value.[1]

The advantage of using mustard over such drugs as cantharides or biniodide of mercury lies mainly in its more rapid, almost instantaneous action, and greater convenience. Nevertheless, I have used both of the last named in the form of ointment, and in a strength varying from 1-20 to 1-40, the former I think too strong, the latter perhaps sometimes too weak. It is possible—and is generally believed—that these two agents have the advantage over mustard in producing more deep-seated effects, and that they cause blisters while mustard is only a rubefacient; but there is no evidence that anything more than a rubefacient is required, or on the other hand that vesication may not be of advantage.

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[1] I am now trying the treatment of a series of chest cases without counter-irritation.

In the present state of our knowledge of therapeutics it is no condemnation of the use of a drug that we do not know how it acts, and for this reason though we have not the least notion how an application of mustard or of cantharides can affect the disease within, this is no bar to its employment, but only a confession of our ignorance.

One argument in favour of counter-irritation—and I now refer almost exclusively to mustard—is that its action can be tested on ourselves, and its beneficial effects, if any, can be recognised. Under these conditions few can fail to recognise its effects in relieving pain, and giving relief to inflammatory symptoms, as for instance, in a neuralgia, a sore throat, or muscular rheumatism, and one is very liable to argue that what suits themselves will suit a similar condition in others. Until all constitutions are made in the one mould such conclusions are illogical and perhaps misleading, but we hold on to them as the nearest approach we can produce to experimental evidence.

All this time I am not losing sight of the fact that what we are considering is whether counter-irritation can be of any service in relieving the congested condition of lung in pneumonia, and perhaps of cutting short the attack by restoring the circulation through the affected area. I can answer the last question first by a direct negative. I do not think that any premonitory pneumonia can be cut short by counter-irritation, but I can express no opinion as to whether the
I am sure, however, that whatever the beneficial action of counter-
lung is in any way relieved by stimulating the skin.
irritation may be, there is no necessity for this action to be severe; a weak mustard application will produce as much benefit as a strong one, while it produces very little pain or discomfort. Anyone disposed to deny this I recommend him, on a favourable opportunity occurring, to try on himself a hot linseed poultice containing one-sixth mustard kept on for twelve hours enveloped in oil-skin, and test this against the mustard leaf of commerce kept on for twelve minutes or as long as can be borne.

If counter-irritation is of any use, I cannot but think that it must be used early. I rarely put on mustard after the second day, and certainly never after the third. I cover the lower half of one or both sides of the chests and also over the heart. If pure mustard be used it is weakened by the addition of from four to six parts of flour, if ordinary domestic mustard be employed two to three parts of flour are mixed with it. This will produce ample œdema of the chest and breast.

If the beneficial action of counter-irritation depends solely on the production of heat in the skin, the same end can be obtained by hot applications to the chest. It is certain that hot applications relieve pain, but there are practical disadvantages connected with fomenting the chest, the chief of which must always be the risk of chill with careless attendants, while prolonged application of a hot rug exhausts the patient, and in the case of pleurisy is said by some to favour effusion. Rubber chest bags filled with hot water are now coming into use, but though I have heard them highly spoken of I have no practical acquaintance with them.

To maintain the skin of the chest-wall at the temperature of the body I have employed a rubber sheet around the patient; it makes the skin wet, and is a simple way of fomenting at a comparatively low temperature, but I abandoned the method after some little trial.

ICE TREATMENT OF PNEUMONIA.

As evidence how the same result may be arrived at by opposite methods may be quoted the ice pack or cold method of treating pneumonia. Bearing in mind how intimately cold and pneumonia are associated, it may appear nothing less than madness to attempt treatment by the application of ice to the chest-wall, but experimental enquiry on the human subject does not support this view, and there are many medical men who regard this method of treatment with the greatest favour, especially, strange to say, in the treatment of infantile pneumonia. Of its action on the horse I know nothing, and it is obvious that there are very few cases where the ordinary practitioner can depart from the generally recognised lines of treatment and experiment on his patients, but it is equally certain some effort should be made to give a new method of treatment every trial. The chief value of the ice treatment in the human subject lies in the reduction of temperature and the relief of restlessness and pain. The latter, it may be observed, is caused by the swelling of the lung and stretching of the pleura.

To sum up what we have said on external applications to the chest, we may say that mustard is probably the most generally useful; it should not be used too strong, and it is probably of no use after the first day or two of the attack. Moist heat certainly gives relief to pain, but is believed to favour effusion in pleurisy, and its application requires more than ordinary care; dry heat in the form of rubber bags appears to be preferable. There is nothing in pneumonia to prohibit even the application of ice to the chest. The object of external treatment would appear to be directed to overcoming the lung congestion and the relief of pain. The latter is certainly accomplished, but of the former there is no evidence, and many practitioners succeed equally well who put nothing around the chest but a dry rug.

TREATMENT OF THE FEVER.

The next line of treatment we have to consider in pneumonia is that directed against the fever and its accompanying derangement of the excretions. For this purpose antipyretics are used, saline laxatives, and diuretics. Even if the laxatives and diuretics employed have no effect on the temperature, they are generally regarded as assisting to get rid of a considerable amount of waste material from the system, the retention of which would be prejudicial. Liq. ammon. acet., magnesium sulphate and potassium nitrate are given with this object, while the high temperature is attacked by quinine.

In human practice attacking pneumonia by combating the temperature is a favourite method of treatment; in other words, the effect is treated rather than the cause, and there would certainly appear to be a few cases where this line of treatment is desirable, but we may at once say that in veterinary practice there is not the slightest necessity for attacking the temperature. It is seldom that it goes so high as to excite any alarm, and it at once commences to fall as the disease begins to exhaust itself. Supposing a temperature rose to 108° and upwards the symptom should be treated accordingly, but under ordinary conditions the treatment of the fever may certainly be neglected.

In the same way the use of mag. sulph., potassium nitrate, etc., is quite unnecessary excepting in very special cases. I have used nothing of the kind for a long time, yet I can remember when I should have considered it tempting Providence to have omitted them. The routine saline method is quite unobjectionable, for the tact of practice dictates that something in the medicine line should be given, and the saline treatment within moderation is harmless. It is believed by some that the saline treatment reduces the strain on the right heart by depleting the vascular system, but I believe that the heart strain in pneumonia is greatly exaggerated, and the reason for this belief will be stated presently.

It has been mentioned earlier in this article that pathologists believe most inflammations can only occur as the result of bacterial activity, and it has been suggested that the fever accompanying inflammation is protective either by inhibiting the growth and production of the bacteria, or by furnishing a larger number of phagocytes for their destruction. Viewed from this standpoint the reduction of temperature by either cooling the surface of the body or by antipyretics would not appear to be sound practice.

It is quite remarkable how high the body temperature may be in pneumonia without producing any alarming symptoms; the equine does not appear to suffer in the nervous system from hyper-pyrexia like the human. Nevertheless, where it is desirable to reduce the body heat, it can be readily done by means of cold water enemata, which I think a better method than wetting the surface of the body; quinine or antipyrin may also be administered, though the latter is not much used in human practice in the treatment of pneumonia.

Chloral has been employed in pneumonia; it lowers temperature and relieves pain. I have used it, but have little experience of its action; theoretically, it would appear to be more useful in pleurisy than in pneumonia. Chloral also slows the heart and dilates the arterioles. Dr Balfour of the Royal Infirmary, Edinburgh,[1] is quite enthusiastic over the action of chloral in pneumonia, and he sums up his views by saying that under chloral treatment patients continue to die, but the mortality is not larger than under the non-chloral treatment. On the other hand the drug confers immense benefit to the patient in the relief of pain, restlessness, cough, the production of sleep, and the lowering of the pulse and temperature.

TREATMENT OF THE HEART.

Incidentally I have referred to the treatment of pneumonia being directed to sustaining and strengthening the heart—keeping it, as the physician terms it, "well in hand." I cannot possibly see the good of thus treating the heart. The behaviour of this organ depends entirely on the intensity of the pneumonia; as this subsides so the pulse drops in frequency and improves in volume, and to direct treatment to the heart looks as if effect were mistaken for cause.

It may of course be that in the human subject, where the heart treatment has such a secure hold, the frequency of a damaged condition of this organ may necessitate special care being paid to it in pneumonia, but this argument will not apply to the horse, which is wonderfully free from heart trouble, and I state without hesitation that as a rule no attention need be directed to the heart, and that the increase in force and reduction in the number of beats will follow subsidence of the pulmonary lesions.

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[1] British Medical Journal, No. 1819, November 1895.

We have previously directed attention to the dilated right heart theory, and the supposed value of blood-letting and saline treatment in reducing the congestion, but an examination of the heart of pneumonic horses does not favour this view; and further, we have seen reason to believe that compensation for lung obstruction occurs in the healthy regions of the organs.

The strongest evidence against what may be termed the heart theory is furnished by those cases which recover. Recovery in pneumonia is more common than death; so soon as a fall in temperature indicates the turning-point in the case, the heart slows down, though the obstruction in the lungs (as determined by auscultation) is infinitely greater than at the early period of the attack, and does not in any degree differ from the obstruction existing at the height of the disease. In other words, the blocking of the lung does not appear in any way to affect the working of the heart, for the lower half of both lungs may be completely solid in a convalescent case, with a heart working rhythmically and powerfully at forty beats per minute.

It appears to me to be impossible to get over this fact, and I would suggest that, valuable as the pulse is in measuring the gravity of the attack, it does not depend upon the extent of obstruction in the lungs, but that heart failure is only one of the effects of the pneumonic poison.

The term poison is used here more as a matter of convenience, for we have previously stated our reasons for believing that the disease in the horse is not a specific infection; still, supposing the view that pneumonia is a specific disease and due to bacterial activity be correct, the production of toxins in the body would fit in more closely with heart failure than does lung obstruction, in which case our treatment would be directed against the toxin rather than against the heart itself.

Considering how helpless we are in the presence of a severe double pneumonia, it is hardly fair perhaps to press too strongly the evidence against heart failure, and so long as pneumonia is being treated empirically those who regard cardiac failure as a cause of death have as much right to their opinion as those who deny it. Heart failure is met in the human subject by the administration of digitalis, or strychnine, or even by the inhalation of oxygen gas. Such methods are equally available in veterinary practice.

THE STIMULANT TREATMENT OF PNEUMONIA.

The stimulant treatment of pneumonia is perhaps the one most commonly employed in veterinary practice; ammonia carbonate, nitric ether, aromatic spirits of ammonia, and alcohol are freely employed. The *rationale* of the treatment is probably that of keeping the heart going, and for many years I employed no other method of internal treatment. It is quite certain, however, that pneumonic cases will do just as well when they receive none of the above drugs, so that in moderation the treatment is harmless. It is well to bear in mind that ammonia carbonate is excreted as urea, and so produces rather a heavy call on the liver and renal epithelium.

We have thus exhausted the routine methods of the internal treatment of pneumonia, and have failed to show that any of the drugs in ordinary use have the slightest effect in controlling the disease. Perhaps this is evidence that pneumonia is a specific affection, in which case we must probably look for its antidote in the serum or other method of animal therapeutics, and in fact the treatment of pneumonia of the human being by means of the serum of an immunised animal is already an accomplished fact; of any attempt to deal with pneumonia of the horse on these lines I have no knowledge.

THE USE OF IODINE IN PNEUMONIA.

Before dealing with what at present must be called the chief method of treating pneumonia, viz., nursing, it is necessary to draw attention to an extensive series of observations on the value of iodine in pneumonia. The use of iodine was suggested by a paper published by Mr Malcolm on the value of iodine in pleurisy. Its employment by me was purely empirical. I know of no action of iodine which even suggested its probable use in the treatment of pneumonia. The result was arrived at by the usual happy-go-lucky method of therapeutics. In discussing this subject subsequently with Mr Malcolm, he informed me that in giving iodine in pleurisy his sole object was to prevent effusion, and that he had no idea of using it in pneumonia, but as there are so few cases of pleurisy uncomplicated by pneumonia, it is evident that should iodine really prove, as I think it will, an agent of some value in the treatment of this disease, the credit must be given to Mr Malcolm.

If pneumonia depends on an organism, it appears to me that the probable action of iodine is as a bactericide. I will show presently that it readily enters the blood, and it is conceivable that in this fluid it acts directly on the organisms producing inflammation. The acceptance of this view does not necessitate the adoption of the theory of a specific disease, as long as pathologists are correct in stating that inflammations are generally the outcome of bacterial activity. I am particular in avoiding the use of the term specific in the action of iodine on inflamed lungs; if iodine possessed a specific action on pneumonia I should expect it to cut short the attack or stay the spread of the disease. It has neither of these actions; the lung inflammation under iodine runs the same course and occupies the same time that it would do were no iodine given, and this no doubt is a gravely weak point in my claim for iodine as a remedial agent, but under the influence of this drug I have had many remarkable recoveries amongst animals which I cannot help but think would otherwise have died. In stating this I have said all I know of the virtue possessed by this drug.

Mr Malcolm recommends the iodine to be given in the drinking water in pleurisy, but I found that very few animals would drink their water when so treated. Thinking that perhaps the beneficial action of the iodine in pleurisy might be due to limiting the amount of fluid consumed, I tried this, both with and without the administration of iodine, but without success in preventing effusion. Further, I came to the conclusion that the amount of iodine contained in the dose recommended by Mr Malcolm was insufficient, and accordingly resorted to the administration in the form of a bolus of the sublimed iodine of the pharmacopœia. This I found to be perfectly soluble in the digestive canal, and it appears in the urine in about twelve hours. This is, of course, undeniable evidence of its absorption into the blood.

It may be urged that the starch in the food of the horse converts the iodine into the insoluble iodide of starch. But iodide of starch is perfectly capable of conversion into sugar by starch-converting ferments, by which means the iodine is liberated, and so far as one can tell none of it is lost; that is, I have never obtained it from the fæces by any of the ordinary tests. In the urine the iodine is excreted as iodide, which may be readily found by adding a little mucilage of starch to the urine and a drop of nitric acid, on the addition of which an immediate blue or purple colour is struck. In the blood serum (employing the inflammatory exudate of pleurisy) I have once found it as iodate and twice as iodide, but more observations are needed. The observations on the urine are very numerous and beyond all doubt.

The dose of iodine depends on the severity of the case; in acute cases of pneumonia and pleuro-pneumonia I administer half-an-ounce of re-sublimed iodine per diem, but in less severe cases two drachms will be found sufficient. I once fancied I detected a slight degree of iodism after continued doses of the drug; but I am not sure of it, and in the very many cases where large doses of iodine have been given for several days I have never observed anything approaching toxic symptoms. If iodism does occur in horses it is very rare; and, as a matter of fact, experiment shows that very much larger doses of iodine than I have recommended may be given to horses with impunity. In my tables will be seen the number of cases of pneumonia and pleuro-pneumonia treated with iodine, and the mortality. I hold no brief on behalf of iodine; I entirely deny, with but one or two exceptions, the specific action of drugs, and it is possible that all the cases which recovered in which iodine was used would have done equally well had no iodine been given. I cannot attempt to dispute this, but I do not think so.

To Mr Malcolm of Birmingham we are indebted for the suggestion of iodine in preventing effusion in pleurisy; but it cannot be too clearly stated that even large doses of iodine from the first moment of the attack may be quite powerless to prevent effusion. I think with Mr Malcolm that the drug does appear to possess some controlling effect, though of course it is not a specific.

Another drug which I have employed is perchloride of iron. It was believed by the late Sir Andrew Clark to check exudation in pneumonia. I have shown in my table the cases in which it was employed, but the number is so few that I hesitate to give an opinion as to its value.

If neither iodine nor iron have been of any use in the treatment of pneumonia, it is equally certain they have done no harm, and such cannot always be said of treatment in general.

NURSING IN PNEUMONIA.

Turning now from special to general treatment, we are, as a profession, quite unanimous as to the absolute necessity of treating lung cases under the best available hygienic condition ; plenty of fresh air, warmed if possible in winter, a large loose-box, clothing, bandages, attention to the bowels, and a nutritious diet are the main points to attend to. For reasons previously given I prefer a sawdust bed, as there is nothing in it to wind around the legs or interfere with movement ; as the horse becomes convalescent, and is inclined to lie down, he may be placed on straw as forming a warmer and more comfortable bed.

The ventilation must be regulated according to the weather—as much fresh air as possible, but no draughts. It would be quite an easy matter to have boxes arranged for pneumonic cases which could be artificially heated by means of gas or oil; and I am sure in the depth of winter they would prove very useful. The employment of sawdust as bedding would at once do away with any risk of fire.

The clothing should not be too heavy, but it should be warm. A hood is an advantage in the depth of winter, and straw or hay bands to the legs during this season will often be found of the greatest value in keeping up the circulation.

The bowels should be attended to. The fæces are sometimes dry, hard, and small ; an enema will be found to rectify any trouble in this direction.

As to diet, anything they like to eat, and within moderation as much as they like. Severe pneumonic cases should always have something by them, little and often ; a large quantity of food placed before them disgusts. I rarely use a manger to feed from, but build up with straw or anything available, a table on which is spread some hay, and on this the feed is placed. Two or three such mangers may be made and used alternately. Conservative as the horse is in a state of health, he is by no means adverse to variety when sick ; change his diet or modify it the instant he shows a disinclination to feed, let him find here and there a little sweet hay, grass, scraped carrots, etc., and in this way a man who has any aptitude for nursing a sick horse will succeed in inducing him to eat sufficient material to keep up his strength.

To keep a horse on a low and non-stimulating diet in order to subdue the inflammatory trouble within, is in pneumonia a pernicious and ignorant practice. I give a liberal nutritious diet, and as much as they can eat. I am sure it is one of the secrets of success.

My table shows how well the appetite is maintained in the pneumonia of young horses ; where it is otherwise I do not hesitate to hand feed, and the best and most convenient material is gruel. This is made sufficiently thin to pour out of a soda water bottle or horn, and this small amount administered every hour causes but little distress, and represents a fair amount of nourishment in the course of the day. I never allow a horse to starve himself, and I do not think with some that food forced on a sick horse does him no good. It is not usual in medical practice to allow a patient to go day after day without nourishment of some kind. Well prepared gruel mixed, if necessary, with milk or eggs is a nutritious and easily digested diet for horses that have to be hand fed.

The fluid drawn off from the chest in pleurisy may, so long as it is aseptic, be used in the treatment of the patient. It can be mixed with gruel and administered, and it may yet be shown to possess antitoxic properties. It is not with this object that I have given it, but to prevent the waste of a highly concentrated and valuable material.[1]

I have known the amount of hay given to be limited, probably under the impression of relieving the lungs from pressure; it is not a practice I have adopted, and when, as is often the case, hay constitutes the bulk of the material they will voluntarily eat, I am sure it is a mistake to limit the amount.

It is unnecessary at the end of the nineteenth century to impress on the practitioner the value of fresh air in pneumonia. I would far rather treat a case in the open than in a dark "fuggy" corner of a stable. Clothing, bandages, hood, and a good bed will always keep the patient warm, a screen could easily be arranged to break the wind, and a few bales of compressed straw make an excellent enclosure. I mention these simple facts to show how readily one can adapt themselves to circumstances, but, as a matter of fact, actual open air treatment is rarely required. In the worst appointed stable there is generally a shed, coach-house, or other place available for a serious case, and this should be appropriated.

There is one point, however, which is not so generally noticed, and that is the benefit arising from a change of scene. Whenever I have a chest case hanging fire I invariably change the box; a complete change is best, but even a change from one side of the stable to another is not to be despised. It is remarkable how such a change is appreciated by the patient.

I generally remove the shoes, but have no fixed rule in this respect.

STATISTICAL AND OTHER DATA.

In the following tables are given the chief features of the majority of the cases of chest inflammation on which this communication is based. Every attempt has been made to render them as accurate as possible, and any doubtful point has been queried. The tables are very complete with the exception of the column showing the subsequent effect on the wind. I have reason to believe that in the majority of the cases where the condition of the wind is queried soundness existed, but as no definite statement exists in my notes I have made no entry.

Many cases have been added to the tables since this paper was written, and this will account for any difference in totals observable between the text and the tables.

In all the tables the fatal cases come last.

The mortality from pneumonia was 11·4 per cent.
„ „ „ pleuro-pneumonia was 37·5 per cent.
„ „ „ pleurisy was 16·66 per cent.
„ „ calculated on the total number of cases was 18 per cent. nearly.

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[1] Professor M'Fadyean informs me that he regards this practice as dangerous, as the fluid from the chest may contain the strangles organism.

[TABLES.

PNEUMONIA.

No.	*Fever period in days.*	*Max. Temp.*	*On what days obtained.*	*Max. Pulsations.*	*Max. Respirations.*	*Time which elapsed before lung symptoms appeared.*	*Which lung affected.*	*How much of each.*	*Treatment.*	*Appetite.*	*Result.*	*Condition of wind after Recovery.*	*Remarks.*[1]
1	11	106·0	1st & 3rd	60	36	2 days	First L then R	½ L ½ R	Iodine	Good	Cured	?	—
2	20	105·0	1st	60	24	—	„	½ L ½ R	„	Poor	„	?	—
3	11	106·0	2nd	60	20	1 day	„	½	No medicine	„	„	?	Influenza.
4	24	104·0	For three weeks between 103-104	62	20	4 days	Both	½	Iron	„	„	Roarer	—
5	6	106·4	1st	60	30	On admission	Left	½	No medicine	Good	„	?	Influenza.
6	7	105·0	2nd	64	20	1 day	Both	¾ L ¾ R	„	Fair	„	Sound	—
7	12	104·0	1st & 7th	72	20	„	„	½ L ¾ R	„	Good	„	?	--
8	17	104·0	3rd & 8th	66	32	On admission	„	¼	Iodine	„	„	?	Strangles.
9	5	105·0	1st, 2nd, & 3rd	72	20	„	Right	½	„	„	„	Sound	—
10	8	105·0	1st, 2nd, & 3rd	60	40	2 days	„	½	„	„	„	?	—
11	10	106·0	1st	70	30	1½ „	Left	⅔	„	Poor	„	?	—
12	9	105·4	1st	60	20	2 „	Right	—	„	Fair	„	?	—
13	10	105·0	2nd to 7th	60	20	3 „	„	½	„	Good	„	Sound	—
14	8	106·0	6th	70	36	1½ „	Left	½	„	„	„	„	—
15	10	105·0	3rd, 4th, & 5th	88	60	3 „	„	⅔	„	Fair	„	„	Influenza.
16	9	105·0	1st	60	20	1 „	Both	?	„	Good	„	„	—
17	13	105·0	1st, 2nd, & 3rd	72	36	1½ „	Right	—	„	„	„	„	—
18	7	103·0	1st & 3rd	60	28	1½ „	Left	½	„	Fair	„	„	—
19	11	105·0	1st to 6th	72	40	2 „	„	⅔	„ in part	„	„	Roarer	before attack.
20	4	105·0	2nd	60	20	2 „	„	½	Iodine	Good	„	?	—
21	6	105·0	1st	60	16	On admission	„	¼	No medicine	?	„	Sound	—
22	6	104·0	1st & 2nd	66	36	1 day	Right	½	Iodine	Fair	„	„	Two attacks
23	14	106·0	1st	60	32	6 days	„	½	„	Good	„	„	Strangles & influenza.
24	14	104·0	1st & 10th	72	36	?	Left	?	No medicine	?	„	?	—
25	7	105·0	1st	60	24	On admission	„	½	?	Good	„	?	Influenza.
26	7	104·0	1st, 3rd, & 4th	60	20	1 day	Both	?	No medicine	?	„	?	„
27	7	106·0	2nd	70	24	On admission	First L then R	½	„	Good	„	?	„
28	15	106·0	1st	84	20	„	Right	¼	„	„	„	?	„
29	9	104·0	1st, 2nd, & 3rd	60	20	„	Left	½	„	„	„	Sound	—
30	?	105·0	?	?	?	4 days	Both	½	Iodine	„	„	?	Strangles.
31	17	105·0	2nd & 4th	60	20	On admission	„	¼	„	„	„	Sound	—
32	?	105·0	?	60	20	4 days	„	½	No medicine	Poor	„	?	Continued fever.
33	?	104·0	?	60	20	10 or 11 days	Left	?	„	„	„	?	„
34	14	104·0	?	70	40	3 days	Both	½ L ? R	„	Good	„	?	Strangles.
35	11	105·0	3rd & 5th	72	20	2 „	Right	?	Iodine for 3 days	Fair	„	?	—

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[1] In this column the word "Strangles," "Influenza," etc., indicate that these diseases existed at or about the same time as the pneumonia.

PNEUMONIA—*continued.*

No.	*Fever period in days.*	*Max. Temp.*	*On what days obtained.*	*Max. Pulsations.*	*Max. Respirations.*	*Time which elapsed before lung symptoms appeared.*	*Which lung affected.*	*How much of each.*	*Treatment.*	*Appetite.*	*Result.*	*Condition of wind after Recovery.*	*Remarks.*
36	?	105·6	?	?	?	On admission	Both	½	Iodine	Good	Cured	Whistler	Complicated influenza case.
37	7	105·0	1st	60	32	2 days	,,	¼	No medicine	Poor	,,	,,	—
38	14	106·0	1st	60	20	3 ,,	,,	L ⅓, R slight	,,	Good	,,	?	—
39	11	105·0	1st & 5th	60	24	1 ,,	,,	⅓	,,	Fair	,,	?	—
40	4	104·0	1st	60	28	On admission	Right	½	,,	Good	,,	?	—
41	4	105·0	1st & 2nd	80	20	1 day	Left	?	,,	,,	,,	?	—
42	24	107·0	1st	66	40	,,	Both	½	Iodine	,,	,,	Sound	—
43	16	104·0	2nd	60	20	?	,,	⅓	,,	,,	,,	?	—
44	9	106·0	5th	60	20	3 days	Left	⅓	,,	,,	,,	?	—
45	5	104·0	1st, 2nd, & 3rd	60	20	3 ,,	First R then L	¼	,,	Poor	,,	Sound	—
46	8	105·0	1st & 2nd	64	28	1 ,,	Both	⅓	,,	,,	,,	?	Two attacks.
47	6	105·0	3rd	60	20	2 ,,	Left	¼	,,	,,	,,	Whistler	—
48	6	106·0	1st	50	20	2 ,,	Right	⅓	,,	,,	,,	?	—
49	7	105·0	1st	50	20	2 ,,	Both	⅓	,,	,,	,,	?	—
50	11	105·0	4th, 5th, & 6th	60	16	1 ,,	,,	¼	,,	Good	,,	Sound	—
51	12	106·0	1st	80	20	3 ,,	Right	½	,,	,,	,,	?	—
52	14	105·0	1st, 2nd, & 6th	60	20	4 ,,	Left	½	,,	Fair	,,	?	—
53	8	105·0	3rd	60	32	2½ ,,	Both	?	,,	Good	,,	Sound	—
54	7	104·4	1st	60	14	On admission	Right	⅓	,,	,,	,,	,,	—
55	8	106·0	1st	60	20	4 days	,,	¼	,,	Poor	,,	?	—
56	8	105·0	1st & 5th	50	20	2 ,,	Both	¼	,,	Good	,,	?	Strangles.
57	13	106·0	2nd	60	12	2 ,,	,,	⅓	,,	,,	,,	Sound	—
58	19	106·0	—	60	20	—	Left	⅓	,,	?	,,	,,	Strangles.
59	16	105·0	2nd	60	20	½ ,,	Both	⅓	,,	Good	,,	?	—
60	14	104·0	—	60	14	On admission	Left	¼	,,	,,	,,	Whistler	Strangles.
61	13	104·0	7th	56	14	,,	Both	⅓	,,	,,	,,	Sound	—
62	18	106·0	2nd	60	14	3rd day	Left	¼	,,	?	,,	,,	—
63	6	106·0	1st	80	40	1½ days	Both	½	,,	Poor	Died	—	—
64	13	106·0	1st	96	44	On admission	First R then L	½	,,	Good	,,	—	Influenza.
65	13	106·0	2nd & 3rd	80	28	1½ days	Right	⅓	?	Bad	,,	—	Strangles.
66	27	106·0	3rd	80	24	?	Both	¼	Iodine	Excellent	,,	—	—
67	15	106·4	5th & 6th	60	20	3rd day	,,	½	No medicine	Bad	,,	—	Strangles.
68	36	105·6	4th & 5th	60	20	1 day	,,	⅓	Iodine	?	,,	—	—
69	13	106·3	1st	60	28	On admission	,,	½	,,	Good	,,	—	—
70	33	106·0	4th	60	30	4th day	First R then L	½	,,	Bad	,,	—	—

PLEURO-PNEUMONIA.

No.	Fever period in days.	Max. Temp.	On what days obtained.	Max. Pulsations.	Max. Respirations.	Time which elapsed before lung symptoms appeared.	Which lung affected.	How much of each.	Treatment.	Appetite.	Result.	Condition of wind after Recovery.	Remarks.
1	7	102·0	6th	72	48	1 day	First R then L	—	Iodine	Excellent	Cured	Whistler	—
2	2	104·0	1st	100	40	On admission	Both	½ L ½ R	Iron	Good	„	„	Strangles.
3	16	105·0	1st & 2nd	80	80	½ day	—	½	Iodine	„	„	Roarer	—
4	6	105·0	1st & 5th	102	80	On admission	Both	?	Iron	„	„	Whistler	—
5	14	105·0	1st, 3rd, 4th & 5th	60	20	1½ days	?	?	„	Poor	„	Sound	—
6	4	105·0	1st	80	30	On admission	L	?	?	?	„	?	Influenza.
7	27	106·0	3rd & 4th	80	40	?	L	?	Iron	Fair	„	Sound	Rheumatism.
8	9	105·0	1st	80	40	On admission	Both	?	„	Good	„	?	„
9	6	103·0	1st	70	40	2 days	L	½	Iodine	„	„	?	Followed by pleurisy.
10	12	105·0	4th, 5th & 6th	70	40	On admission	B	—	„	„	„	?	Died from tetanus.
11	4	106·4	1st	84	40	„	Both	½	„	„	„	?	—
12	8	107·0	3rd	60	16	„	R	⅓	„	Fair	„	Sound	—
13	7	105·0	2nd	64	28	„	L	⅓	„	„	„	„	—
14	8	104·4	1st	64	20	1 day	B	¼	„	Good	„	?	Strangles.
15	29	106·0	4th & 6th	72	14	4 „	L	½	„	„	„	Sound	„
16	18	105·0	2nd, 3rd, & 11th	96	48	On admission	Both	½	„	Poor	Died	—	Chest tapped 17th day.
17	3	107·0	2nd	100	44	„	Both	⅓	Iron	Bad	„	—	Effusion.
18	18	104·0	5th	96	40	„	R	⅓	Iodine	„	„	—	Chest tapped 15th day.
19	19	105·0	9th, 10th, 13th, 14th, & 15th	80	40	„	B	?	„	?	„	—	Chest tapped 7th day.
20	8	104·6	3rd	96	48	„	L	⅓	?	?	„	—	Effusion.
21	10	106·0	1st & 9th	80	60	1 day	R lung first affected then left on 4th day	⅓	Iron	Ravenous	„	—	„
22	4	108·0	1st	108	40	On admission	Both	⅓	Iodine	Bad	„	—	„
23	18	105·0	5th	60	28	4 days	First R then L	⅓	„	Fair	„	—	No effusion.
24	13	105·6	10th	72	20	On admission	B	⅓	„	Good	„	—	Effusion.

PLEURISY.

No.	Fever period in days.	Max. Temp.	On what days obtained.	Max. Pulsations.	Max. Respirations.	Time which elapsed before pleuritic symptoms appeared.	Which side affected.	Treatment.	Appetite.	Result.	Condition of wind after Recovery.	Remarks.
1	4	105·0	1st	72	20	On admission	Both	Iodine	Good	Cured	—	—
2	3	103·2	2nd	60	16	1 day	Left	„	„	„	—	—
3	7	105·0	1st	60	20	„	Right	Iron	„	„	—	—
4	21	105·0	1st & 2nd	80	24	On admission	Left	Iodine	„	„	—	—
5	13	106·0	3rd	80	32	1 day	„	„	Fair	„	—	—
6	5	103·6	1st	60	20	On admission	Right	?	Good	„	—	—
7	12	107·0	1st	96	60	„	?	Iodine	„	„	—	Chest tapped 7th day.
8	12	103·4	1st	80	36	6 hours	First R, L on 2nd day	„	Fair	„	—	—
9	3	103·0	1st	60	14	On admission	Left	„	„	„	—	—
10	8	106·0	4th	60	20	4 days	„	„	?	„	—	Followed strangles.
11	15	106·0	1st	100	40	On admission	Both	„	Good	Died	—	Chest tapped 18th day.
12	18	105·0	—	80	34	„	„	„	„	„	—	Chest tapped 15th day.

THE ETIOLOGY OF EPIZOOTIC ABORTION.[1]

By Professor Dr B. BANG, Copenhagen.

IN most civilised countries epizootic abortion of the cow appears to play a highly important rôle; indeed, in many regions it appears to be the disease which more than any other detracts from the profit of cattle breeding. It is therefore matter for surprise that the disease has, as yet, been to such a slight extent accurately investigated.

Although it must be admitted that abortion may arise from the most various causes (sporadic abortion), and even assume enzootic proportions when such causal circumstances operate simultaneously on a large number of pregnant animals, yet, at the present day, it can hardly be denied that the disease by far most frequently occurs in circumstances that compel us to assume the existence of an infectious agent.

As early as the beginning of the present century this view found expression in England. For example, in the widely read book "The Complete Farmer," 5th edition, 1807, the following occurs: "It is considered certainly contagious, and when it happens the abortion should be immediately buried, and the cow kept as widely apart as possible from the herd, and not receive the bull that goes with them."[2] With respect to this point it may be remarked that there is a resemblance between the disease now under consideration and tuberculosis. As the result of simple observation the layman had very early recognised the infectious nature of the disease, but the representative of science hesitated to adopt this view; at least this was the case towards the middle of the present century,[3] and it was not till the beginning of the bacteriological era that the contagious nature of abortion again gradually came to be generally recognised. This theory was very strongly advanced by Zundel[4] and Styrl,[5] and very important contributions to the elucidation of the question were made by Franck, Roloff, Brauer and Johne.

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[2] Penberthy. "Enzootic Abortion." Journal of Comparative Pathology and Therapeutics, Vol. VIII., page 100, 1895.

[3] Hurtrel d'Arboval writes in 1826: "One is not content with the belief that it is epizootic, for it is also said to be contagious, but in that another erroneous view has been put forward." (Dictionnaire I., page 122, Avortement.)

Youatt: "There is no contagion, but the result is as fatal as the direst contagion would have made it. ("Cattle," 1834, page 529.)

Rychner: "As regards the causes of abortion, a predisposition is often present, and is very frequently inherited, and hence it comes that epizootic influences tend to produce abortion among many animals." Infection is not even mentioned. (Bujatrik, 1841, page 179.)

Hering writes: "The ordinary causes are not accurately known; there are years in which abortion occur so frequently and with such a wide distribution in one or several species of animals that it cannot be explained on the ground of accidental and local causes." And he adds thereto: "Tögl and Tessier write regarding epizootic abortion, and the latter indeed names it contagious." (Pathologie, 3rd edition, 1858, page 690).

As late as in 1878, Stockfleth warns against the absolute acceptance of the idea of contagion. He says, "Such a theory is, no doubt, quite convenient, as it makes us overlook other more essential causes, which it may be very difficult to detect and to remedy, but it may thus do much harm." The apparent contagion he ascribes to a pollution of the air in the byre. (D. Zeitschrift für Thiermed., Bd. IV., p. 177.)

[4] Recueil, 1871. In speaking of the causes, however, he distinguishes those which may originate enzootic abortion and those which spread it, and says: "Enzootic abortion is in fact propagated by contagion."

[5] Traité d'obstetrique vétérinaire, 1875. Under the head of "De l'avortement epizootique,' one finds a very clear critical exposition of the etiological hypotheses, and several valuable observations by French veterinary surgeons which make the infectious character of the disease probable.

The first, and as yet the only one, to submit the question to accurate investigation was Nocard, who, in December 1885, was commissioned by the Minister for Agriculture to study the epizootic abortion which had for years prevailed among the excellent breeds of cattle in Nièvre.

In the general consideration of the question with which Nocard introduces his report,[1] he mentions that up till that time there had been very few detailed descriptions of the condition of the fœtal membranes and fluids, and none at all regarding the uterine mucous membrane; and he makes the very weighty observation that this admits of very easy explanation, since abortion never directly causes death, but is fatal only through the secondary inflammation of the uterus which is set up by the retention of the placenta, and then the alterations which were perhaps the cause of the abortion are difficult to distinguish from those which have set in as the result of sepsis of the fœtal membranes. Strictly speaking, all that was known was that "as a rule the after-birth is much altered, macerated, and covered with pus, and that the cow that has aborted furnishes for a long time afterwards a foul, purulent discharge."

After having established the infectious nature of the disease on the basis of admirable clinical observations, he discusses the various hypotheses which might be brought forward in explanation of the anatomical changes causing the abortion. The first, namely, that the epizootic abortion might result from a general affection of the mother, may, he thinks, be put aside, first, because the cow both immediately before and immediately after the act of abortion exhibits no symptoms of disease, and second, because there is no elevation of temperature, and it is impossible to recognise either by macro- or microscopic examination, or by bacteriological investigation, any changes in the urine, blood, or milk, or in the various tissues. It therefore only remains to examine whether abortion is the consequence of a *local affection of the genital organs of the mother*, or whether it is a disease of the fœtus or its envelopes.

Although at the *post-mortem* examination of cows which had quite recently aborted, he found between the uterus and the fœtal envelopes a considerable quantity of yellowish flocculent purulent material, he nevertheless came to the conclusion that it was not a case of disease of the uterine mucous membrane, because, in sections made through the uterine walls, he could recognise only an intense epithelial desquamation. On the contrary, he thought that he was able to make out from his investigations that epizootic abortion is brought about by a bacterial disease of the fœtus and its envelopes.

His bacteriological investigations are naturally of most interest to us. In the puriform substance deposited between the uterus and the fœtal envelopes, Nocard found two different microbes abundantly present, namely (1) micrococci, isolated, in pairs, or in short chains of from three to five members; and (2) short, thick bacilli, isolated or united together in pairs. Both could be stained with methylene blue. The micrococci were most abundant in the fluid lying at the base of the cotyledons and round about them, while the bacilli were by far the most abundant in the juice expressed from the follicles of the cotyledons.

In order to cultivate these bacteria he unfortunately did not use a separation method, but only simple stab cultures in peptone gelatine or bouillon. After forty-eight hours the latter medium had become turbid. When the inoculation was made from cotyledon juice the bouillon contained, it was said, a pure culture of the short bacilli, while when the inoculation was made from the yellow flocculent material the cultures contained both the bacilli and the micrococci. A growth showed itself in gelatine also. After inoculation with cotyledon juice fine parallel streaks grew outwards from the needle track towards the sides, and when the inoculation was made from the yellowish material small whitish, round, granular colonies formed along the line of perforation.

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[1] Recueil de Médecine Vétérinaire, 1886, p. 669.

In one case he had the opportunity to examine a fœtus that was still contained within the uterus. He made inoculations from the amniotic fluid in bouillon, and thus obtained a culture of micrococci, which he held to be analogous to the micrococci above mentioned.

His examination of aborted fœtuses (which had died immediately) did not reveal any microbes in the blood, bile, serous fluids, spleen, liver, kidneys, thymus gland, or lymphatic glands. On the contrary, he found in the intestinal contents in all cases (fourteen) a quantity of different microbes (*microbes variés*). Furthermore, when he inoculated bouillon and peptone gelatine from the medulla oblongata he obtained a growth of micrococci, which, it was said, were very similar to those above mentioned. The idea of making this peculiar examination had been suggested to him by Roux, owing to the analogy between the peculiar continuous bellowing of aborted calves and the altered voice of a rabid dog.

Nocard also makes the observation that aborted fœtuses putrefy with striking rapidity, and, in his opinion, this stood in connection with the constant presence of microbes in the intestine.

The recognition of two different sorts of bacteria is by itself calculated to raise a certain amount of doubt regarding the causal importance of these, and, as a matter of fact, he did not succeed in leading decisive proof on this point—that is to say, cause pregnant cows to abort by inoculating them with one of the microbes which he had discovered.

The cause of Nocard's non-success is ascribable to the following: The main cause of it undoubtedly was that he was not in possession of an appropriate nutritive medium, since he did not add blood serum to the substances which he employed. Besides that, the material which he used for examination was not quite free from objection. Most of the cows had aborted some time before the examination of the uterus; the best of them was slaughtered eight and a half hours after the expulsion of the fœtus, within which time foreign bacteria might easily have penetrated, and the majority of the fœtuses had been expelled one to two days before they were examined. In only one case had he the opportunity to examine the yet unopened uterus of a pregnant cow immediately after slaughter (the only material absolutely free from objection), and unfortunately this case appears to have been not at all an appropriate one. He alleges, it is true, that the signs of impending abortion were in this cow unmistakable, but it is certainly remarkable that it was just in this case, in contradistinction to all the others, that he was unable to recognise any flocculent, purulent secretion between the uterus and the fœtal envelopes. It is therefore in my opinion extremely doubtful whether it was in reality a case of epizootic abortion.[1]

Although it thus appears that Nocard's investigations did not lead to a conclusive result, there still remains to him the great credit of having collected good clinical observations in support of the infectious nature of abortion, and also of having pointed out the right way in which to find the cause of the disease.

The investigations which I shall now describe were carried out in my laboratory by myself and my assistant, Mr V. Stribolt, veterinary surgeon. The latter has had a very essential share in them, and all the experiments bearing on the peculiar behaviour of the abortion bacillus with regard to oxygen were carried out by him.

As soon as we had placed before ourselves the task of discovering the cause of epizootic abortion, it became evident that we must obtain a pregnant cow from a herd suffering from the disease, and indeed we required such a cow already showing the signs of impending abortion. In this we easily succeeded, thanks to the friendly assistance of Veterinary-Surgeon Fasting, in Frederiksborg, who was able to indicate in his practice a large farm on which many cows had aborted. From this herd we purchased on the 19th of December, 1895, a five-year-old cow, which had been served with the bull on the 21st of May, and which since the 15th of December had shown the well-known premonitory symptoms of abortion. The cow was there and then slaughtered under Mr Stribolt's directions, and after the genital organs had been removed from the body he bound an elastic ligature round the vagina and brought the preparation to the laboratory, where it was examined six hours after slaughter.

The external surface of the uterus was normal. The os uteri was firmly closed and the cervical canal was filled with the normal thick mucus. After disinfection of the serous covering of the uterus by burning, I made a section through the uterine wall; when the mucous membrane was divided we saw *between that and the fœtal envelopes an abundant odourless exudate*—a dirty yellow, somewhat thin, pultaceous material, of a slimy, somewhat lumpy character. At some places where the fluid constituents had run out the exudate was of a semi-solid nature; its reaction was alkaline. When it was allowed to stand in a glass it separated into two strata, namely, superiorly a reddish-yellow cloudy serum, and at the bottom a thick greyish-yellow precipitate.

On cutting through the chorion, we saw under that a thin, clear, apparently gelatinous substance, with very fine membranes running through it; closer examination showed that this was the fine connective tissue lying between the chorion and the allantois, saturated with an œdematous exudate. This was present over the entire extent of the fœtal envelopes, and formed a layer 1½ cm. thick. The allantoic fluid was natural in appearance, thin, yellowish, and containing only fine flocculi. Nothing abnormal was observable in connection with the amniotic fluid. The umbilical cord was œdematous. The size of the fœtus and the degree of development of the hair on it indicated an age of seven months. It was quite fresh, and on section it showed no striking alteration. The pericardium contained a little reddish fluid; the intestinal mucous membrane was, perhaps, rather redder than ordinary; the spleen was in very slight degree swollen, and the blood was fluid.

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[1] From the case here mentioned he obtained a culture of his short bacillus after inoculation from cotyledon juice, and of the micrococci after inoculation from liquor amnii. In the intestine of the fœtus he found a quantity of microbes, and in cultures made from the medulla oblongata micrococci made their appearance.

The examination of a cover-glass preparation made from the yellowish exudate and stained with Lœffler's methylene-blue immediately showed the presence of a very small bacterium, apparently in pure culture. This organism was present in very considerable numbers; many examples lay free, but most striking were the large dense clumps of bacteria. Closer examination showed that these heaps were included within cells, whose bodies were often in this way greatly distended. Sometimes the body of the cell was very indistinct, but as a rule one could still recognise external to the heap a part of the cell body, and often also the cell nucleus. Not seldom the cell body had assumed a peculiar homogeneous appearance.

In the dense heaps the bacteria mostly had the appearance of cocci, but some of the free-lying individuals were of a longer shape, and these were at first regarded as short oval structures; closer examination, however, under very high magnification showed that we had in fact to deal with a small bacillus,[1] whose body contained one, two, or more rarely three, roundish or elongated granules. These granules most readily took up the stain. The length of the bacilli is very variable; the largest examples are about as long as tubercle bacilli.

The before-mentioned granules frequently lie at the ends of the bacillus, but they may occur a little distance from the extremities. They stain with the ordinary anilin dyes, but not by the method of Gram. The bacilli are non-motile. In the subchorial œdema I found no bacteria. In the heart blood of the fœtus there were a few, and in the intestinal contents there were many staining granules, but it was not possible to say with certainty whether these were bacteria or not.

This discovery indicates that the *epizootic abortion ought to be regarded as a specific uterine catarrh, determined by a definite species of bacterium.* It is true that the uterine mucous membrane was not strikingly altered, but a chronic catarrh is not necessarily associated with striking anatomical alteration. In my opinion the very abundant exudate, which contained a quantity of shed epithelial cells, pus cells, and detritus, must necessarily have been furnished by the uterine mucous membrane, and not by the thin chorion, and consequently the disease must be regarded as a uterine catarrh.

In the fœtal envelopes the œdematous condition of the fine subchorial connective tissue represented the most striking change. This œdema ought probably to be ascribed to a state of stagnation in the fœtal circulation. Curiously enough, this condition is not distinctly mentioned in the literature with which I am acquainted, although, according to my observations, it is constantly present. In all the fœtal membranes that I have seen from cases of epizootic abortion, it was present to a more or less pronounced degree, and it is to practical veterinary surgeons a well-known fact that the after-birth of a cow that has aborted is uncommonly thick, slimy, and gelatinous, and that the removal of it is difficult, owing to the ease with which the membranes tear.[1]

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[1] This is most easily recognised in preparations from a bouillon-serum culture. With fuchsin staining the whole body of the bacillus often takes on the colour (*see* Fig. 4).

The presence of the uterine exudate is also a well-known fact, but one has not deduced from it with sufficient distinctness the seat and nature of the disease. Most practical veterinary surgeons know that during, or immediately after, the act of abortion, or sometimes even before it, a more or less abundant, dirty, reddish-yellow, slimy, flocculent, pus-like fluid escapes from the vagina, and that this is easily distinguished by its odourless character and its appearance *immediately after the birth* from the discharge which results from an ordinary uterine inflammation. When the after-birth is retained the discharge, of course, even after abortion, gradually becomes offensive in smell in consequence of the penetration of putrefactive bacteria.

After this short digression I return to our first case. The next object in our investigation of the disease, namely, to cultivate the bacterium which we had found in a state of purity, and to furnish evidence that it constituted one definite species, was very easily accomplished by making cultures in test tubes containing serum-gelatine-agar[2] and placing them in the incubator. At the end of two days in the case of the first tube, and two days after that in the case of the third (the second tube was accidentally broken), there appeared a great number of *very small colonies, which developed only in a definite zone of the tubes.* This zone lay about ½ cm. under the surface of the nutritive medium, and it had a thickness of from 1 to 1½ cm.; colonies were not present either above or below this. We had thus not to do with an ærobic bacterium, which would have pushed its growth as far as the surface of the nutritive medium, and still less had we to do with an anærobic form, which would have grown as far as the bottom of the tube. The under limit of the zone of growth lay exactly where the upper limit of the growth of a strictly anærobic bacterium shows itself (the necrosis bacillus, for example). This highly peculiar behaviour of the abortion bacillus towards oxygen made it at once apparent that we had to do with a distinct species, and therefore rendered it in the highest degree probable that there was a causal connection between the bacterium and the disease.

Obviously, the peculiar mode of growth which has just been mentioned greatly facilitated our further studies, as we were thereby enabled to recognise the specific bacillus even when it was present in very impure cultures.

The colonies are, as has been mentioned, very small, often punctiform, the largest scarcely attaining to the size of a pin's head. Their form is roundish, and when slightly magnified their edge is seen to be finely dentated. They are composed of small bacilli similar in appearance to those found in the uterine exudate.[1]

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[1] Not seldom one finds the fœtal membranes somewhat œdematous after normal parturition, but scarcely to such a degree as after abortion.

[2] This nutritive medium (originally introduced by Stribolt) is very useful, especially for the investigation of anærobic bacteria. After having liquefied the agar-gelatine (that is to say, ¾ per cent. agar and 5 per cent. gelatine) the liquid is cooled to 45° C, and it is then mixed with about half its volume of liquid sterile serum. One now sows some of the material in the first test tube, and in the ordinary way makes several degrees of dilution, pouring a few drops from the first into the second tube, and from that into the third, and so on. The test tubes are then immediately cooled and solidified in a stream of water. This mixture of serum and gelatine-agar remains beautifully transparent, and permits accurate observation of the isolated developing colonies, as well as the starting of fresh cultures from them. The solid medium also serves very well for the preparation of stab cultures by means of a glass needle. Hereafter, for the sake of brevity, I call this nutritive medium simply agar-serum.

We tried, of course, to obtain cultures from the uterine exudate in other nutrient media, and in other conditions. No growth took place in gelatine-agar, either by the method of plate culture or when the medium was left in the test tubes. In strict correspondence with the above-mentioned sensibility of the abortion bacillus to the oxygen present in the atmospheric air, we found that no growth took place at the surface of solid serum or when the agar-serum was poured out on plates. In glycerine bouillon (5 per cent), however, we succeeded in obtaining a very sparing growth, that is to say, after about fourteen days we saw a very scanty fine precipitate, which contained a few small whitish granules. These granules contained bacilli similar in appearance to the abortion bacilli, and when reinoculated in agar-serum they grew in the typical manner. In pure liquid serum the bacillus also grows only sparingly, but it thrives somewhat better in a mixture of serum (one part) and glycerine bouillon (two parts).

I shall now mention some investigations which still further explain the *peculiar behaviour of the abortion bacillus towards oxygen.*

As was to be expected, one does not succeed in cultivating the bacillus when the oxygen is removed by alkaline pyrogallol solution. The before-mentioned mode of growth of the bacillus in a definite zone under the surface of the agar-serum appears to show distinctly that the organism requires oxygen, but does not agree with such a large quantity of it as is present in atmospheric air, for otherwise it would grow in the very uppermost strata, and even at the free surface of the medium itself. It is therefore all the more striking that Stribolt succeeded in altering in an important degree the conditions of growth of the bacillus, by replacing the atmospheric air above the agar-serum as far as possible by oxygen. He sowed the bacilli in fluid agar-serum, which he then poured into small quadrangular flasks (Nielsen's culture flasks), in which the medium formed a thin stratum on one of the walls. He then conducted oxygen (90 per cent., as it occurs in commerce) into the flasks, and afterwards closed the neck of the flask by means of melted paraffin. *The bacilli now grew very actively in the thin layer of agar-serum as well as at its surface.* He also succeeded in rendering the growth of the bacilli in glycerine-bouillon-serum distinctly more luxuriant by bubbling a large quantity of oxygen through the fluid, and then closing the neck of the flask with paraffin. In these conditions the medium became diffusely turbid, and a somewhat rich, finely granular precipitate of abortion bacilli was formed.[2]

From this one might suppose that it is perhaps the carbonic acid present in the atmospheric air which interferes with the growth of the abortion bacillus at the surface, and in the uppermost strata of the agar-serum. This hypothesis, however, is contradicted by the fact that the addition of from 4 to 5 per cent. of carbonic acid to the oxygen did not affect the result in the above-mentioned experiment. Moreover, the removal of the carbonic acid from the atmospheric air by inserting a cotton wool plug saturated with caustic soda solution in the upper part of the test tubes, had no influence on the growth of the bacillus (the possibility that the nitrogen here played a part is considered below and contradicted).

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[1] It ought to be added that small air bubbles not infrequently make their appearance in the nutritive medium, and not exclusively in the zone of growth. They are especially numerous in the first tube when a large amount of material has been made used to inoculate it, and they may then occur throughout the whole of the medium.

[2] The growth in bouillon-serum, however, was somewhat variable, and sometimes it was very slight or did not take place at all. No doubt this depends upon the greater or lesser tension of the oxygen (*see* below).

Strange as it may appear, we must in the meanwhile conclude from the above-mentioned experiment that the abortion bacillus cannot bear the presence of oxygen in the concentration of 21 per cent. (as it occurs in atmospheric air), but that, on the other hand, the presence of an atmosphere very rich in oxygen exerts a very stimulating influence on the organism.

The following experiments[1] appear to show beyond any doubt that *for the abortion bacillus in its behaviour towards oxygen there are two optima*, namely: first, a degree of oxygen tension in the nutritive medium less than that of the atmospheric air; and second, the presence in the nutritive medium of a very high oxygen tension, which, however, lies somewhat under 100 per cent. Between these two optima there is an intermediate zone, in which the abortion bacillus grows badly or not at all.

When one displaces the atmospheric air above the agar-serum in a test tube by oxygen (as may be done by conducting the oxygen through a fine glass tube inserted through the cotton wool plug, and then closing with melted paraffin, or by placing an agar-serum culture tube in a litre flask and filling the latter with oxygen through the rubber stopper), the zone of growth assumes the following appearance: Under the surface there lies a stratum in which, with the naked eye, one discovers no colonies or extremely few (with a slight magnification one sometimes finds several examples).[2] Then follows with a sharp upper boundary a stratum which has taken a dull whitish-grey colour owing to the fact that it is filled with very numerous and relatively large colonies. In the downward direction this stratum (the upper optimum) becomes gradually clearer because the colonies are less compressed and also somewhat smaller. Hereon follows a rather broad stratum in which, with the naked eye, one discovers only a few colonies; after that one comes suddenly to a stratum which is sharply defined and closely beset with medium-sized colonies (the lower optimum). In this stratum the colonies do not attain to the same size as in the upper layer. This zone of growth is gradually lost in the downward direction, until all growth ceases. That the intermediate zone is never quite free from colonies, may, we think, be explained by the fact that the oxygen can only penetrate gradually into the nutritive medium, and hence at every point of the intermediate zone there must at a certain moment exist just that tension of oxygen which corresponds to the conditions in the lower optimum; at that moment the bacilli there present find the conditions favourable for their development, but subsequently the oxygen tension becomes too great, and then the growth stops. That the optima zones are less sharply defined below than above no doubt depends upon the gradual penetration of the oxygen.

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[1] These experiments were carried out mainly at the instigation of the physiologist, Dr Henriquez, and we are indebted to him for many valuable hints regarding the method of conducting them.

[2] The absence of growth at the surface is to some extent opposed to what was found in the previously mentioned experiments in which plate cultures were made in a flask filled with oxygen, since in these the colonies occurred even at the surface. To explain this one must suppose that in these experiments the oxygen tension was less.

The following experiment was made in order to show that the above-mentioned conditions of growth are not influenced by the nitrogen present in the atmospheric air. Test tubes or glasses of agar-serum were inoculated and placed in an iron vessel, which was then filled with atmospheric air under a tension of five atmospheres. In these conditions the partial pressure of the oxygen should be almost exactly the same as that of pure oxygen, and if the nitrogen has no influence on the growth of the abortion bacillus one would expect to find that the above-described mode of growth would be repeated; such we found to be the case. The photographic picture reproduced in Fig. 2, illustrates the conditions very distinctly.

It must, however, be mentioned that one does not always succeed in bringing out the before-described conditions of growth so distinctly. When, for example, the pressure in the iron vessel is not kept continually at the same height (as happened on one occasion through a leak in the stopcock), then, of course, both the optima do not appear distinctly. A greater or smaller addition of serum to the gelatine-agar mixture also appears to have a certain influence. Lastly, it appears to be of importance whether one, in adding the serum, shakes the fluid much or little, because with strong shaking more air is absorbed. In one case, for example, the serum and the agar-gelatine were mixed in a flask, then sucked up with pipettes, and distributed in the tubes, instead of as usual making the mixture in each separate tube. Probably through this procedure more than an ordinary amount of air was taken up, and perhaps that is the explanation why in this case the cultures, which were placed in a flask filled with oxygen, showed an upper optimum zone, while under that zone the whole tube (for a height of over 3 inches) contained colonies, which, however, were very small.

Lastly, it ought to be mentioned that when the air over the nutritive medium is much rarefied, as happens when the culture tube is placed in a small flask out of which part of the air is pumped, the zone of growth reaches to the surface. When the rarefaction is continued until the pressure amounts to 2 inches of mercury all growth stops.

Through the kindness of the botanical physiologist, Mr W. Johannsen, our attention was called to the fact that for the higher plants also a condition of affairs analogous to that here recognised for a bacterium has been alleged to exist. For example, A Wieler[1] found that the growth of several plants was accelerated when they were put under bell jars which were filled with rarefied air. With a certain degree of rarefaction he found an optimum of growth; with further rarefaction the rate of growth diminished, until it ceased altogether with a complete absence of oxygen. When the plants were put into a gas mixture containing more oxygen than the atmospheric air he thought that he could recognise a retardation of growth as soon as the partial pressure of oxygen corresponded to an air compression of from two to two-and-a-half atmospheres. With a higher partial pressure an acceleration of growth again took place (second optimum), until ultimately with a sufficiently high oxygen pressure the growth stopped altogether. Subsequent investigations by S. Jentys,[2] however, have not confirmed Wieler's views regarding the conditions of growth under a high oxygen pressure. At the same time he was able to recognise in

[1] Pfeffer: Untersuchungen aus dem botan. Institut zu Tübingen, Band I., page 189.

[2] Pfeffer: Untersuchungen, Band II., page 419.

the case of some plants a decidedly favourable influence on growth through increase of the partial oxygen pressure up to one atmosphere.[1]

In 1893, Dr M. W. Beyerinck,[2] in the case of various motile bacteria recognised conditions which have not a little resemblance to the peculiar mode of growth of the abortion bacillus. He describes his principal experiments in the following terms: "One lays a brown bean at the bottom of a test tube, fills the tube nearly quite full with distilled water, and allows it to stand at rest at room temperature. . . . The dry bean immediately becomes swollen up with water, and speedily absorbs the oxygen dissolved in the liquid, while at the same time there diffuse from it certain soluble bodies which serve as nutriment for bacteria. . . . During the swelling of the bean the dried bacteria on its surface spring into activity, and immediately begin to multiply at the cost of the before-mentioned nutritive materials. There thus develops at the surface of the bean a slight turbidity due to the presence of motile bacteria, but this turbidity soon recedes in consequence of the deficiency of oxygen which sets in there. One observes after twenty-four hours, at a temperature of 20° C., or somewhat later at a lower temperature, the following peculiar appearances. Whereas the water in the upper part of the tube remains perfectly clear, a space which is also quite clear gradually develops in the neighbourhood of the bean, since the bacteria accumulated there recede further and further. *At a definite place*, which depends upon the time of the experiment, *the bacteria collect together in an extremely thin stratum* which when regarded from the side appears as a *sharply defined white line.* This stratum I have named a "bacterial level" (Bacterienniveau). With the before-described plan of experiment it is from 2 to 3 cm. distant from the bean after forty-eight hours. He alleges that the bacterial stratum always contained one definite bacillus (Bacillus perlibratus) when he used a bean just taken out of its pod. "The 'level' also marks that place where the oxygen coming from above and the diffusion stream of nutrient matters ascending from the bean meet one another, *and here only do the bacteria find the conditions appropriate for their growth.*" The "level" may remain for several days (it remained apparent for weeks or months by using pure cultures of the bacillus grown on gelatine, of which a small quantity was poured out into the bottom of the tube). It gradually ascends until ultimately a position of equilibrium is reached. When he conducted hydrogen into the test tubes over water the 'level' rose. to the surface in a few hours, and if he then readmitted the air it again sank to that point where the bacteria found the condition most favourable for their nourishment and respiration. If oxygen is then introduced the "level" sinks still deeper.

For various other bacteria he found that similar conditions existed, but that the height of the "level" varied, which fact he ascribes to the varying requirements of different bacteria for oxygen.

Even in a comparatively thin stratum of water included between a glass slide and a cover glass slightly tilted at one edge, he saw some similar figures (respiration figures), and these were different according as he used ærobic or anærobic bacteria, or such as required only a limited supply of oxygen.

After we had succeeded in one case of epizootic abortion in detecting as the probable cause of the disease a peculiar bacillus, we naturally wished to examine a number of other cases of the same sort. Through the kindness of several of our colleagues in the neighbourhood we succeeded in the course of the following months in obtaining suitable specimens from twenty-one cases of epizoötic abortion, namely, part of the after-birth which had been manually removed immediately after parturition, and in many cases also some of the exudate that had escaped from the uterus. In a couple of cases we obtained at the same time the aborted fœtus. During the past winter we have again obtained many such specimens from different colleagues. In all the cases we were able to recognise alterations which corresponded exactly with those observed in our first case. The uterine exudate had a quite similar appearance, save that it was often somewhat reddened owing to its being mixed with a little blood. In the fœtal membranes one could recognise more or less distinctly (often *very* distinctly) the subchorial œdema. The small vessels of the chorion were always more or less injected, and as a rule flakes of pus were adherent to the outer surface of the chorion, while these were always present between the villi of the cotyledons. The appearance of the placental cotyledons was somewhat variable; most frequently they were strongly injected, not seldom hæmorrhagic. They were often of a dirty greyish-yellow colour, seldom, however, throughout their whole extent; more frequently, in part only, and then either in the centre or at the edge while the remaining parts of the same cotyledons were red in colour. All the cotyledons were seldom of a greyish-yellow colour.[1] In cover-glass preparations made from the uterine exudate (or from the pus adherent to the chorion) we could in almost all cases detect the abortion bacilli, partly lying free and partly collected together in intracellular heaps. The number of bacilli appears to be very variable; in some preparations they were immediately apparent, while in others they were very difficult to find. As a rule they are most easily found when the exudate contains very small greyish-white granules, which one may then pick up with a needle. It need hardly be said that such preparations contained not only the abortion bacillus, but always at the same time many other bacteria of various kinds which had penetrated after opening the uterus. Nevertheless, from the great majority of the specimens which reached us in the cold season of the year we were able to isolate the characteristic abortion bacilli by means of separation cultures in agar-serum, and also to obtain pure cultures of the same. In the cases in which the fœtus was forwarded we obtained at once from the intestinal contents pure cultures of the abortion bacillus without a trace of foreign bacteria. It is thus proved that *the abortion bacillus may infect the fœtus.* Probably this may sometimes occur through the medium of the amniotic fluid. In one case in which culture media were inoculated from the blood of the fœtus we obtained no growth, and in the other we obtained immediately a pure culture of the abortion bacilli from the blood and also from the medulla oblongata, fourth stomach, and intestinal contents; this fœtus was five months old.

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[1] From the older investigations regarding the influence of compressed air on the growth of plants I may cite those made by P. Bert (La pression barométrique, 1878). He found that germination was delayed in air compressed to two atmospheres, or in gas mixtures with 40 per cent. of oxygen. With an air pressure of five atmospheres the interference with the process of germination was very marked (cited by Jentys, *loc. cit.*) In the case of anthrax bacilli, as is well known, Chauveau in 1884 observed an attenuation of the virulence as a result of the action of compressed oxygen on cultures, and in this way he prepared a vaccin (*see* Nocard and Leclainche: Les maladies microbiennes des animaux, page 169).

[2] Uber Athmungsfiguren beweglicher Bakterien. Centralblatt für Bakteriologie und Parasitenkunde, Bd. XIV., page 827.

We have also had the opportunity to recognise the abortion bacilli in the uterus in two cases of dead mummified fœtus. On the 16th of March 1896, at the slaughter-house here, I made the *post-*

[1] In these cases the fœtus had been dead before abortion.

mortem examination of a four-and-a-half years old cow which formed one of the reacting division of animals at Thurebylille (the farm at which I have carried on the main experiments with tuberculin). This cow had calved in October 1893 and then aborted in September 1894, about two months before the date of parturition. Subsequently the cow apparently would not conceive and she was therefore fattened. In the uterus of this cow I found a mummified fœtus about 9 inches long and closely invested by the fœtal membranes. This shrunken fœtus was surrounded by a large quantity of brownish, thick, extremely tough tenacious exudate. As I was aware that epizootic abortion had prevailed in this stock for years, it occurred to me to examine the before-described exudate for the presence of the abortion bacilli. In cover-glass preparations I was unable to detect the bacilli, but a pure culture of these developed in agar-serum; the colonies, however, were unusually slow in developing, since they did not appear distinctly until the ninth day. They were also rather few in number. *It is thus seen that the uterine catarrh which is set up by the abortion bacilli does not always entail the expulsion of the fœtus, but that the result is sometimes merely the death of the fœtus.* When the latter is contained in the uterus one can easily understand that a large quantity of exudate gradually forms around the shrunken envelopes which invest a mummified fœtus, and that this exudate itself in the course of time becomes inspissated and takes on the before-described tough, tenacious consistence.

Subsequently we had a quite similar case. On the 15th of February 1897, Mr Stribolt made a *post-mortem* examination of a heifer rather more than two years old, which had been sent in from the sound division at Thurebylille. This animal had been bulled on the 19th of March 1896; at the beginning of September while at grass it showed the premonitory symptoms of abortion, but these gradually disappeared when the animal was brought into the house. At the *post-mortem* of the animal, which was otherwise sound, Stribolt found in the uterus lesions exactly corresponding with those which I have described above, save that the mummified fœtus was somewhat smaller. In cover-glass preparations made from the yellowish-brown tough exudate bacilli could not be detected, but agar-serum tubes inoculated from the same yielded a pure culture of the abortion bacillus after six days. In this case also the number of colonies was very small. It is thus certainly permissible to draw the conclusion that in those cases in which one finds in the uterus a mummified calf surrounded by a large quantity of yellowish-brown, tough, tenacious exudate, the death of the fœtus has been caused by the specific inflammation of abortion; whereas the death of the fœtus must be ascribed to other circumstances (*e.g.*, torsion of the uterus) when a mummified fœtus is closely surrounded by the uterine wall.[1]

The before-described cases are also interesting inasmuch as they illustrate the great vitality of the abortion bacillus. The first cow had probably been bulled in December 1894, or in January 1895, and the death of the fœtus had probably taken place five months later, that is to say, in May or June 1895. The bacilli had therefore remained for at least nine months longer in the uterus, and, nevertheless, they were alive. In the second case the death of the fœtus had occurred in September 1896, and the bacilli were still alive five months afterwards. We are also able to prove the vitality of the abortion bacillus in another way. At the *post-mortem* examination of our first case in December 1895, we collected some of the uterine exudate in sterile test tubes, which were placed in the ice chest. On the 8th March, 13th May, and 14th June 1896 we inoculated agar-serum from these tubes, and in each case obtained a perfectly pure culture. Hence, under these conditions the bacilli retain their vitality for at least seven months.

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[1] At the *post-mortem* examination of a Thurebylille three-year-old cow, in the year 1893, I found what probably represents an early stage of this affection. The cow had aborted on the 29th of November 1892; when she was slaughtered on the 7th of July 1893 I found in the uterus a large somewhat macerated fœtus, surrounded by dirty reddish membranes, while between them and the uterine wall there was a large quantity of turbid fluid. The fact that in this case the fœtus was not mummified and the uterine exudate not inspissated, is probably explained by the shorter interval which had elapsed between the death of the fœtus and the slaughter of the cow.

This property of the abortion bacillus is certainly of the very greatest importance in connection with the disease of which it is the cause. It makes it easy to understand how a cow that has once aborted has a great tendency to abort again during the following pregnancy, since the bacilli remain in the uterus unless a careful disinfection of the uterine cavity is carried out. The vitality of the bacillus also explains the difficulty which is encountered in getting rid of the disease from an infected building.

The above-described investigations made it highly probable that we had actually discovered the specific cause of epizootic abortion, but in order to make the proof complete it was necessary for us to bring about abortion by introducing abortion bacilli into the bodies of pregnant cows. As is well known, several veterinary surgeons have already reported that they have succeeded in doing this by introducing vaginal secretion or part of the after-birth of aborted cows into the vagina of pregnant cows. It is also extremely likely that the agent of infection is, as a rule, taken in in a similar way, the external parts of the genital organs of the cow being brought into contact with infected objects during her sojourn in the byre. When the bull conveys the disease one must assume that infection takes place during copulation. It therefore naturally occurred to us to carry out inoculation experiments by simply introducing pure cultures of the abortion bacillus into the vagina. Our first experiments in that direction, however, proved unfruitful, for the two cows, into the vagina of each of which pure cultures of the bacillus had been introduced, during the following nineteen and twenty-nine days respectively showed no sign of impending abortion, and when they were slaughtered after these intervals we could not detect the slightest pathological change in the uterus or in the fœtal envelopes. However, the result was the same in the case of two other cows into the vagina of each of which we introduced part of the after-birth from cows that had aborted. When these two cows were killed, thirty-three and thirty-five days respectively afterwards, we found no lesions. We were greatly surprised at these results, since experiments carried out in the same way by Brauer were said to have been followed by positive results in from nine to twenty-one days, those by Lehnert after twelve to twenty days, and those by Trinchera after nine to thirteen days. The previous history of the cows used for our experiments was not known, as they had been sold for slaughter in the

Copenhagen cattle market. It was therefore conceivable that they might have come from infected stocks, and have repeatedly aborted and thereby acquired an immunity. We thought at first that this hypothesis might explain the non-success of our experiments. I have, however, since observed that English experimenters in similar experiments have in some cases found a much longer period of incubation, viz., five-and-a-half to ten weeks.[1]

It therefore appeared quite reasonable to suppose that our experimental cows would have aborted if we had not slaughtered them so soon, and that we did not find the slightest trace of uterine inflammation might perhaps be explained by supposing that the bacilli had not yet penetrated into the uterine cavity.

Experiments I. and II. In order to guard against the possibility of an immunity accidentally existing in the experimental animals, we now purchased two pregnant cows from a stock in which abortion was unknown. One of the cows was four years old, and the other seven years old, and they had been bulled on the 14th and 16th of January respectively. These cows were placed in one of the horse stables in my division of the clinique, and on the 14th April 1896 we injected into the vagina of each a rather large quantity of pure culture of the abortion bacillus, the culture being partly in glycerine-bouillon-serum, and partly in agar-serum. The seven-year-old cow received cultures that had been grown in an oxygen atmosphere, and the other one cultures which had been made in contact with ordinary atmospheric air. By the aid of a large glass pipette the cultures were introduced as far as the anterior part of the vagina. Inasmuch as the cows exhibited no pathological symptoms during the first five weeks, we feared that these experiments were also going to miscarry, and we therefore repeated the injections on the 23rd of May and 4th of June.

At length, on the 23rd of June we observed in the seven-year-old cow a relaxation of the pelvic ligaments, an œdematous swelling of the labia of the vulva, and a slight flow of mucus from the vagina. The four-year-old cow on the same day showed a slight œdema of the lower parts of the labia of the vulva, but the pelvic ligaments were still firm. Nevertheless the latter cow aborted at 4 A.M. on the 24th June. The fœtus was five months old, and it had apparently been dead for some days, as the claws were loose and the cornea opaque, while an abundant exudation of reddish serum had taken place into the subcutaneous tissue and into the thoracic and abdominal cavities. The after-birth was retained, and when it was partially detached at 9 A.M. we observed in it a very abundant subchorial œdema. The chorion was much injected, the cotyledons of a dirty yellow colour, and between the villi of the latter there lay a large quantity of a muco-purulent flaky exudate, which was also discharged from the uterus in moderate quantity. The exudate contained the abortion bacilli, but other bacteria, namely micrococci, had already found their way into it, although the examination was made six hours after the act of abortion.[1] The cultures were thus impure, but by means of separation methods of culture in agar-serum we were able to recognise the colonies of the abortion bacillus in the usual stratum.

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[1] Compare "Second report of a Committee appointed by the Highland and Agricultural Society of Scotland" (7th April 1886), consisting of Dr G. Sims Woodhead and Dr A. P. Aitken, to whom were added Professor M'Fadyean, and more recently Mr Campbell, F.R.C.V.S., Kirkcudbright, "Journal of Comparative Pathology and Therapeutics," Vol. II., page 97.

We had the seven-year-old cow slaughtered on the forenoon of the 24th June, in order that we might have the opportunity to examine the still unopened uterus. From a small incision in the latter we obtained a little exudate, in which we again found the abortion bacilli, and from which we immediately obtained pure cultures of the same in agar-serum. The uterus was then laid on ice in order that I might have the opportunity to demonstrate the specimen five days later on the occasion of a lecture at a meeting of the Veterinary Union. On that occasion I was able to recognise quite the same alterations as I have already described in connection with our first observation (page 128), but the subchorial œdema was still more striking than in that case. The fœtus was quite fresh, and it had therefore beyond doubt been alive when the cow was slaughtered.

By both these experiments we have furnished the complete proof that the bacillus discovered by us is the cause of epizootic abortion. They also prove that the mere deposition of the bacilli in the vagina can produce the disease. The organism must thus be able to propagate through the mucus of the cervical canal. Unfortunately the experiments do not furnish any certain information regarding the period of incubation, owing to the fact that three separate attempts to infect were made. In my opinion, however, it is most probable that the first inoculation was the active one, and that the period of incubation in these cases was therefore ten weeks.

Since the publication of my lecture[2] we have had the opportunity to pursue our experiments regarding the production of abortion by inoculating with cultures of our abortion bacillus. I shall describe in the first place an experiment with a cow.

Experiment III. This was a six-year-old animal affected with chronic pyelo-nephritis, which I had purchased in October 1896 for the purpose of another experiment. Having accidentally discovered that the cow was pregnant, on the 19th January we introduced into her vagina, in the same way as in the two above-mentioned cases, a culture which had been grown in oxygen. On the 9th April (that is to say, after eighty days) the cow gave birth to a small but apparently full-term calf, which at first appeared healthy, but after about fourteen days was attacked with diarrhœa, and had to be killed. At the time of parturition there escaped from the uterus a not specially abundant, reddish, muco-purulent, flaky exudate, which contained the abortion bacilli. The after-birth, which was somewhat œdematous, was retained, and this induced a rather severe attack of metritis, which however ended in recovery. In this case the abortion bacilli had thus brought about the specific uterine catarrh, and it is also probable that in consequence of this parturition had set in a little too soon, but the pathological alterations developed so slowly that the duration of pregnancy was comparatively little influenced. Although this case speaks in favour of a long period of incubation, I must admit that even it does not justify us in drawing such a conclusion, inasmuch as we had practised a subcutaneous injection of abortion bacilli on the 6th of March. On that date we injected into the neck of the cow about 18 cc. of a bouillon-serum culture; in consequence of this injection the cow had a sharp attack of fever lasting for three days, but at the seat of injection there formed only a small and but slightly sensitive infiltration, which entirely disappeared after a few days. The injection was made on the 6th of March at 3 P.M., and the following is the temperature record:—

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[1] Compare Nocard's experience, who found various species of bacteria when he made the examination eight and a half hours after abortion.

[2] Maanedskrift for Dyrlager, 8 Bind, p. 146: August 1896.

6th March, 3.30 P.M.	38·9°	7th March. 5 P.M.	41·3°
„ 5.30 „	39·4°	„ 7 „	41·1°
„ 7.30 „	39·9°	„ 12 „	40·6°
„ 10 „	40·4°	8th March, 4 A.M.	40·6°
„ 12 „	40·6°	„ 7 „	40·0°
7th March, 2 A.M.	40·8°	„ 12 „	40·0°
„ 4 „	41·2°	„ 7 P.M.	40·8°
„ 6 „	40·6°	9th March, 10 A.M.	40·3°
„ 8 „	40·2°	„ 7 „	40·7°
„ 11 „	41·1°	10th March, 10 A.M.	39·1°
„ 2 P.M.	40·7°		

In this experiment we had in view merely to quite provisionally obtain information as to whether by inoculation with cultures or with the toxin obtained from the bacillus one could obtain a diagnostic indication regarding the presence of the abortion inflammation in pregnant cows, and we, of course, intended in later experiments to inject sterile cultures. The marked increase of temperature, which was also accompanied by slight loss of appetite and the passage of soft fæces, appeared to indicate the possibility of a favourable result. However, on the 8th of March we practised a subcutaneous injection to a healthy bull calf, and this showed that the reaction fever can have no diagnostic importance, for in this animal a quite similar elevation of temperature took place and lasted for about four days.

So far as is known to me very little accurate information regarding epizootic abortion among sheep is to be found in the literature. It is generally said that these animals are less inclined to abort than cows; and the cause of abortion in them is usually ascribed to general injuries (sudden change of food, over-exertion, fright, etc.) According to Franck,[1] however, abortion on a large scale is not infrequently observed in flocks of sheep, and he is inclined to attribute the same to infection by straw impregnated with septic material. I have heard from Danish veterinary surgeons and farmers that abortion among sheep has been observed at places where epizootic abortion prevailed among cows, and the investigations of the already mentioned Scottish

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[1] Geburtshülfe, 1876, page 276.

Commission showed that the introduction of vaginal discharge from an aborted cow into the vagina of a pregnant sheep could produce abortion. We therefore determined to make some experiments with sheep also. Unfortunately, our experimental animals were already well advanced in pregnancy.

Experiments IV. and V. On the 29th January 1897, we inoculated intravaginally (as in the cows) two sheep, namely, a three-year-old white ewe, which had been tupped on the 7th of November, and a two-year-old black ewe, which had been tupped on the 11th of November. The first received about 20 cc. of an agar-serum culture grown in oxygen, and the black ewe the same quantity of culture which had been grown in atmospheric air.

On the 3rd of April, that is to say 147 days after copulation, and hence probably a little too soon, the white ewe gave birth to two living lambs, which throve quite well. The cotyledons of the after-birth, which soon came away, were very red, and on them, as well as on the surface of the much injected chorion, there was a not inconsiderable quantity of thick, reddish, turbid exudate, which contained an immense number of the abortion bacilli, partly lying free, and partly included within cells. In agar-serum they grew in the customary manner. This experiment therefore shows that the abortion bacilli, even in sheep, can penetrate from the vagina into the uterus, and there set up a specific catarrh. If the sheep had been inoculated at an earlier period of pregnancy it is very probable that a decidedly premature parturition would have set in. The incubation period in this case was sixty-four days.

The black ewe was killed on the 3rd of April. The result in this animal was negative. The uterus contained two large lambs, and on the outer surface of the envelopes there was present only a very small quantity of a whitish, slimy fluid, in which neither by microscopic examination nor by culture in agar-serum could we detect bacteria. This experiment certainly proves that abortion bacilli introduced into the vagina do not always penetrate into the uterus.

Experiment VI.—It now appeared to us to be of interest to determine experimentally whether it was possible to introduce abortion bacilli into the uterus by way of the blood stream. On the 6th of March 1897, we therefore injected into the jugular vein of a pregnant ewe 18 cc. of a bouillon-serum culture.[1] On the following day the sheep would not feed, respiration was very hurried, and it had a temperature of 40·9° C. On the 8th of March the respiration was still somewhat hurried, but the animal was otherwise better; temperature 39·5°, no swelling in the neck. As early as the 18th of March the ewe gave birth to two lambs, which were small but healthy, and developed quite well. The after-birth came away after an hour; the entire chorion and also the cotyledons were deep red, and covered with a rich red, flaky exudate. The membranes were a little œdematous. The exudate contained very large numbers of abortion bacilli, of which we obtained beautiful cultures.

Experiment VII.—On the 1st of April 1897, we injected 8 cc. of a bouillon-serum culture, which had been grown in oxygen, into the jugular vein of a small ewe, whose time for lambing was not known.

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[1] In the opinion of the seller the ewe was due to lamb at the beginning of April.

The animal belonged to a flock which had been used by my colleague, C. O. Jensen, for braxy inoculations, and in consequence of this inoculation it had a large wound on one hind leg. It was somewhat sickly, and therefore the immediate consequences of the experiment were not so pronounced. The fever on the second day was not important, but on the third day the temperature was 41° C. On the 8th of April it gave birth to a small living lamb, weighing 1840 grammes. The after-birth was covered by a sparing quantity of turbid exudate, which contained abortion bacilli as well as a few other bacteria. The sheep died on the 11th of April, in consequence of a not recent, diffuse, sero-fibrinous, double pleurisy and peritonitis. In the exudate I found immense numbers of streptococci which in all probability had penetrated at the skin wound.

Although in this case it may possibly be doubted whether the termination of the pregnancy was accelerated by the injection, nevertheless both these cases prove that *the abortion bacilli can be introduced into the uterus through the blood stream also.* Experiment VI. also shows distinctly that the introduction of abortion bacilli into the blood excites a transient fever (just as after subcutaneous inoculation), but no essential lesions in the internal organs. Only in the mucous membrane of the pregnant uterus does the organism find the conditions favourable for its development, and there only does it set up a specific inflammation.

A knowledge of the possibility of the penetration of the abortion bacilli through the blood stream is also of interest, in so far as it raises the question whether or not the agents of infection may sometimes be taken in through the respiratory or digestive organs.[1] I certainly do not doubt that, as a rule, the sexual organs form the port of entrance. There are cases, however, in which it is difficult to see how infection can take place in this way. I refer especially to those cases where on large farms the heifers are kept in separate byres, isolated from the cow byres; where the heifers are served by a young bull that has never been in connection with cows that have aborted; and where, nevertheless, a not inconsiderable number of the heifers abort. In such cases it is easy to suppose that the heifers, for example, have been infected while grazing in fields where cows have aborted.[1]

We have also taken the opportunity to investigate the influence of the abortion bacilli on mares.

Experiment VIII.—On the 1st of April 1897 we injected into the jugular vein of an eight-years-old pregnant mare about 25 cc. of a bouillon-serum culture which had been grown in oxygen. One hour afterwards the temperature was 37·7°, but in six hours it had risen to 40·2°. On the following day at noon the temperature was 39·4°, and in the evening 39·2°; on the succeeding morning 38·1°. Notwithstanding this transient elevation of temperature the mare showed no symptom of disease, and the appetite was good. On the 29th of April she gave birth to a small living foal, which was strikingly weak, took little nourishment, and breathed quickly. It died on the 1st of May, and at the *post-mortem* examination I found the lungs very incompletely inflated (fœtal atelectasis). The after-birth began to come away after three hours, and it was with some difficulty removed. The chorion was perhaps a little redder than usual, and on its outer surface there lay a considerable quantity of a reddish-grey, mucopurulent, flaky exudate. There was a little œdema in the subchorial tissue, and a larger quantity between the allantois and the amnion. The exudate contained masses of abortion bacilli, and by inoculating agar serum we obtained beautiful cultures with the typical appearance.

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[1] Nocard has already called attention to this possibility.

This case is in complete agreement with the sheep experiments, only here it was beyond doubt that the birth was premature, and although from a single experiment one ought not to draw the conclusion that epizootic abortion among mares[1] is always determined by the bacillus discovered by us, that is nevertheless in the highest degree probable; at any rate one ought not to entirely neglect the possibility of the infection taking place from cows and sheep to mares.

We have not yet had the opportunity to make any experiments with goats. I have, however, been informed by Mr Fasting that a goat which had been placed in a cow byre where abortion prevailed (in consequence of an old superstition that this would protect the cows against abortion) twice aborted about seven weeks before full term, and exhibited a discharge similar to that seen in cows. Still another colleague observed a case of abortion in a goat which was kept in a stock where the cows had formerly aborted. It is hence in the highest degree probable that our bacillus is infectious for goats.

It appears to be a well established fact that although a cow that has aborted shows a great tendency to abort again at the next pregnancy, and perhaps even the third year if then pregnant, nevertheless this tendency gradually diminishes, so that it is quite exceptional for the cow to continue aborting year after year.[2] In correspondence with this, it is the rule that outbreaks of abortion spontaneously disappear after some years, provided that new cows are not being constantly brought in. It has been alleged by several observers that in each successive case the period of pregnancy at which abortion takes place becomes later and later.[3] Whether this actually ought to be regarded as a rule must in the meantime be left undecided. In the discussion of these questions (so far as I can see) we lack a solid basis of sufficiently numerous accurate observations, and are therefore exposed to the risk of drawing too far-reaching conclusions from single observations and insufficiently controlled estimates. In the hope that it may serve as an incentive to the collection and publication of accurate statistical data from as many affected herds as possible, I will here record the experience in connection with the farm of Thurebylille.

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[1] In the year 1751, at the breeding stud at Frederiksborg, 51 out of 208 mares (24·5 per cent.) aborted; in the year 1762, 31 out of 163 (19 per cent.) aborted, whereas the percentage of abortion among mares was ordinarily only 3·9 (Prosch: "Hestens Avl og Pleje," 1870, S. 200). According to Penberthy, at the present day epizootic abortion occurs very frequently in England and North America, sometimes to an alarming extent, so that in large studs the most of the mares are affected (*loc. cit.*, pages 98 to 107).

[2] Compare Sand's views founded on the reports from Danish veterinary surgeons (Deutsche Zeitschrift für Thiermedicin. 21 Band., page 204).

[3] Zundel. Recueil, 1871, page 167. Penberthy says that while some cows which have once aborted become sterile, and others usually abort again, there is a tendency for the period of gestation to be prolonged after each succeeding conception, and that after a while (three to five years) the disease in a herd into which no fresh cows are introduced tends to die out, as with specific contagious diseases, owing to acquired immunity of the cows themselves, or to attenuation of the virus (*loc. cit.*, page 104). Compare also Sand, who repeats the opinion of several veterinary surgeons, that when abortion has prevailed for two or three years in a herd only the heifers and the cows that have been bought in abort. Nocard (Recueil, 1886, page 675) mentions the retardation of the date of abortion in a byre as a common symptom, though not a constant one (n'a rien de fixe ni de regulier). He does not, however, expressly state that it is the same cows that abort later the next year. According to his account cows that have once aborted are, as a rule, prepared for the butcher. If the retardation thus applies to other individuals the cause of that would appear to be an *attenuation of the virulence* (see under).

The stock of cattle consists of a little over 200 head, of which 130 are milch cows and thirty heifers. Since the spring of 1885 (in which year the management was taken over by the present company) an accurate account has been kept of the circumstances connected with pregnancy, etc. The number of aborting animals has been as follows: 16, 11, 13, 18, 13, 15, 26, 20, 23, 14, 22, 17. And here a peculiar fact shows itself, namely, that whereas during the first year no primiparæ aborted, the number of these that subsequently aborted increased in the following manner: 1, 4, 9, 8, 10, 16, 11, 13, 7, 11, and 9. Thus, out of the total number of 208 animals that aborted, ninety-nine, or about the half, were primiparæ, and in some years about two-thirds were primiparæ. This corresponds well with Sand's views on the subject, and I have also heard from several Danish veterinary surgeons and farmers that a similar state of affairs is of common occurrence on the larger farms, where it is chiefly the heifers and the in-bought cows that abort; the latter, however, as a rule aborting first on the year following purchase, inasmuch as they are usually bought in the pregnant condition, and the bacillus therefore has not the necessary time to penetrate into the uterus.[1]

I have in the next place examined into the later history of the primiparæ which have aborted their first calf. In this inquiry I have of course to leave out of account the nine which have aborted for the first time during last winter. Of the others, five were slaughtered soon after abortion, and in the case of two no record has been preserved. *Of the remaining eighty-three only twenty have aborted during the following year*, and seven did not become pregnant. Five out of these seven were slaughtered, and two calved at full term during the third year. *Fifty-four animals that aborted as primiparæ carried their calves to full term during the following year*, and thirty-one of these have also calved at full term for many years in succession afterwards, while others have after one or several normal parturitions proved barren, in which case they were as a rule fattened and slaughtered. Only eight of the fifty-four animals have subsequently after one (five cases), two (two cases), or three (one case) normal parturitions again aborted, most of them only once, but one animal two years in succession. Of the twenty animals that aborted both their first calves, two carried their third calf for the full term, but aborted the fourth again. Four animals have also aborted the third calf; these were then fattened and slaughtered, and this was also done with the remaining fourteen.

In the older writings it is asserted that secundiparæ (cows carrying their second calf) are especially apt to abort, and a physiological explanation of this has been sought in the excessive strain which is put upon such young animals.[2] On Thurebylille, however, the number of young cows which have gone to full term during their first pregnancy but have cast their second calf, is in no way great, namely, only sixteen. Of these cases two occurred last winter, and of one there is no record; of the remaining thirteen, six were slaughtered soon after, two were barren in the following year, three aborted during the following year (one of these also the year after that), while one subsequently went three times to full term, and another six times to full term.

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[1] Nocard has already called attention to this fact, and says: "On peut en enduire que la maladie doit avoir *une assez longue* période d'incubation." As an example he mentions a farmer who took over the stock on a farm where abortion had been very prevalent, and introduced into this stock thirty-two cows at from the sixth to the eighth month of pregancy. All these cows calved at full time, but the next year eleven of them aborted at from the fourth to the eighth month (*loc. cit.*, p. 674).

[2] Stockfleth d. Zeitschrift für Theirmedicin. 4 B., page 172.

Of the multiparous cows whose first abortion occurred later than their first pregnancy, I find that six aborted two years in succession, and one three years in succession; two again aborted once or twice, with intervening normal parturitions.

I thus find that out of all the animals at Thurebylille only thirty cows have aborted in two successive years, and of these only six have cast their calves three times in succession; twenty-two cows that had aborted would not conceive during the following year, but eighty-one cows carried their calves to full term. These figures are not in accordance with the commonly held view that a cow which has aborted is very apt to repeat the abortion during the following year or years.[1]

I will not deny the correctness of that view, but I believe that on closer examination it will be found that it applies especially to outbreaks in previously uncontaminated herds. Under such circumstances it may be the rule that the cows first acquire a relative or absolute immunity after they have aborted two or three (or several) times. In large herds, such as are common on our farms, where the plague very frequently continues in existence for many years (probably often for over fifty years), the *virulence of the disease appears to become attenuated*, so that in the case of many animals one abortion serves to confer a temporary or permanent immunity. In this way one may, I think, explain the above-mentioned fact regarding the aborting heifers at Thurebylille. It may be that in such herds there is a congenital indisposition. Such a view is supported by a number of veterinary surgeons, who allege that on large farms the bought-in cows are more apt to abort than those which have been bred on the place.

The fact that at Thurebylille abortion in very many cases takes place at a very late period of pregnancy speaks in favour of an attenuation of the virulence. Among the cases in which the exact period of abortion is correctly recorded, that occurred in thirteen cases one month or less before the completion of full term; in thirty-seven cases between one and two months, in twenty-seven between two and three months, and in only twenty-six more than three months, before full term.

The experience at Thurebylille does not confirm the above-mentioned view regarding a retardation in the case of repeated abortion. Only in six cases did the second abortion set in at a later period of pregnancy than the first; in ten cases the cow cast the second calf (sometimes even the third) at the same or an earlier period than the first.

The account here given of the course of the plague in a large herd in which it was stationary for years is in my opinion of great interest, inasmuch as the facts are well calculated to divert one's thoughts from the view of infection. When the abortion as a rule affects only the heifers carrying their first calf and in-bought cows, and when in the majority of cases the abortion is not repeated, one is inclined to seek the cause in entirely different conditions, such as conception at too early a period of life, change of food, chills on damp cold pastures, etc.—in fact, in the great arsenal of suggested causes of abortion which from time immemorial have been found in the text-books. I do not doubt that more accurate investigation will show that the majority of cases of "enzootic" abortion (Johne), or "plague-like" abortion (Harms), the cause of which has hitherto been sought in ordinary defects of feeding, general management, etc., will actually be found to belong to *bacterial abortion.* In this connection I may point out that when, for example, Stockfleth, in opposition to the theory of infection, cited the fact that cows often abort while at pasture, this argument at once falls to the ground when one remembers that the period of incubation is often, and indeed probably as a rule, a long one, so that such animals may very well have been infected while they were kept indoors.

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[1] Nocard states: "Bien rares sont les vaches qui ayant une premiere fois avorté parcourent heuresement toutes les phases de la gestation et mettent bas à terme un veau bien conformé" (Recueil 1886, page 677).

In considering the way in which infection takes place too little importance has hitherto been attached to the bull. Nocard, for example, does not mention the possibility of the bull being the carrier of the contagion. Penberthy, on the contrary, speaks of this as *primâ facie* probable, and also says that now and again proof of this method of infection is present. Trinchera and Reindl have also given expression to similar views, and we are indebted to Sand for having collected several confirmatory observations from Danish veterinary surgeons. It is indeed self evident that if the vagina is to be regarded as the chief port of entrance of the contagion, copulation must afford the most favourable opportunity of all for infection, as that is the only way in which a direct introduction of the virus into the uterus can be effected. The specific abortion catarrh affords a close analogy with the gonorrhœal inflammation of the mucous membrane, and on *a priori* grounds one is inclined to apply this analogy also to the mode of infection. That this has not hitherto been done as much as it deserves to be no doubt depends upon the fact that people have hitherto been inclined to assume that the period of incubation is a short one. When, however, we know that the abortion catarrh may be, and probably as a rule is, insidious in its course, we must recognise the great probability of infection being spread by means of the bull. It is in the meantime not possible to pronounce a certain opinion as to how often infection takes place by means of the bull, but I am inclined to think that it is of frequent occurrence. Besides those already mentioned by Sand, I know of several similar observations made in Denmark,[1] and the best of these I shall cite hereafter. Mr Winsnes, veterinary surgeon in Christiania, has also informed me that on several farms in an isolated position in Norway, and on which no bull was kept, he has seen abortion break out after cows had been taken to the bull at neighbouring farms where the plague prevailed. It is of very great practical importance to keep this circumstance in mind, and I would here call attention to the risk of spreading the disease which is associated with the custom, otherwise so useful for the improvement of the breed (and very general in Denmark), of keeping a bull to serve the cows of all the members of a society (*Stiervereinen*).

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[1] Compare the discussion after my lecture on the 29th of June 1896. Maanedskrift for Dyrlaeger, Bd. VIII., page 165.

In this connection I must still touch upon the question as to whether it is possible that a cow that has calved at full term may nevertheless furnish a vaginal discharge that is infective. In my opinion the possibility must certainly be admitted when one remembers that from several quarters it has been alleged that aborting cows have at each succeeding pregnancy a tendency to abort at a later period.[1] Furthermore, Mr Paulsen, veterinary surgeon, Kingsted, alleges that in herds in which abortion prevails it may often be observed that the after-birth of cows that have carried their calves to full term shows morbid alterations, being œdematous and covered with exudate, as is common in animals that have actually aborted.[2] Such animals must be capable of infecting others.

By way of conclusion I may make some observations regarding the prophylactic treatment of epizootic abortion, but in that I can be brief. It is evident that a recognition of the long-suspected agent of infection must in the highest degree inspire confidence in the now generally recommended treatment which is based on the principles of isolation and disinfection. It is precisely this increased confidence in rational prophylactic treatment that I regard as the most valuable acquisition for practice in the meantime. It will be generally admitted that such treatment is capable of yielding very good results, and, if nevertheless, the evil has scarcely diminished, that is no doubt due to the fact that this method of treatment has not as a rule been vigorously and consistently carried out.

So long as we did not actually know the contagium and its way of entrance into the body, and could merely form conjectures, we cannot wonder that the farmer has not generally recognised the necessity of taking immediate measures against infection, or that he has frequently at a later stage grudged the necessary amount of trouble, and the not inconsiderable expense. Now that we are able to tell him that abortion belongs to the category of infectious diseases, and are able to explain to him that the agent of infection is in the uterus and first gains the outer world when the fœtus is expelled, that it afterwards may persist for months in the genital organs of the cow and probably for just as long in the floor of the byre and on objects soiled with the vaginal discharges, that it is actually capable of penetrating from the vagina into the pregnant uterus, that the bull may be an active agent in the dissemination of the disease, etc., he will be more inclined to follow the advice of the veterinary surgeon.

The discovery of the abortion bacillus will, it may be hoped, exert on the rational prophylactic treatment of abortion a stimulating influence similar to that which the discovery of the tubercle bacillus did in the case of tuberculosis. Before that discovery it was known that tuberculosis was an infectious disease, and methods of treatment grounded on that belief were recommended, but the discovery of the bacillus and an accurate knowledge of its conditions of existence first pointed a way to the successful struggle with tuberculosis. As an example of how much may be done by adopting a rational method of prophylactic treatment against epizootic abortion, I will cite the following observation, for which I am indebted to a young colleague, Mr M. Paulsen, veterinary surgeon, Ringsted. A farmer who owned sixteen cows, among which abortion had never occurred, nine years ago allowed seven of his cows to be served by the bull at a neighbouring farm, where abortion had prevailed for some years. When the first of these cows began to show symptoms of impending abortion she was immediately removed from the byre, and that was also done with the other six when they in succession exhibited the same symptoms. In the whole seven cows the after-birth was retained, and Mr Paulsen therefore had it removed by hand within twenty-four hours. The removal was complete in the case of six of the cows, but not in the seventh, which became emaciated and was soon afterwards sold. After removal of the after-birth the cows were for some time washed out daily with carbolised water, and they were not readmitted to the byre until some considerable time afterwards, when they appeared to be entirely free from discharge. During the following year the six cows again became pregnant and carried their calves to full term. No case of abortion has since then occurred in this herd.[1]

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[1] Zundel says; "If the animals are well fed the period of gestation lasts longer, and the cow which has aborted at the sixth month will abort the second time at the seventh, and then a little before the ninth, so that after three years she will carry her calf to full term." (Recueil, 1871, page 467.)

[2] Maanedskrift for Dyrlaeger. Band. VIII., page 169. A farmer among whose cows abortion has prevailed has also made a similar observation. He observed in several cows that calved at the normal period a discharge the same as that with which he was familiar in aborted cows.

This observation furnishes a good example of the importance of the bull as a carrier of the contagion, and an excellent model of prophylactic treatment, Since the cows were isolated as soon as they showed symptoms of impending abortion no abortion bacilli were discharged into the byre. The diseased animals were first readmitted after complete disinfection of their genital organs, and this disinfection was also successful in putting an end to the disease in them.

In cases in which aborting cows have not been removed from the byre before abortion it is obvious that one must carry out a careful disinfection of the byre (especially of the stalls of the cows, the gutter behind them, and all the utensils that may have in any way become infected). The fœtus and its envelopes must be buried or burnt, and if possible the cow must be removed to another building, and attended by some person who is prohibited from entering the byre. When many cows abort, as is especially the case on large farms, it is unfortunately often impossible, at least in northern countries during the winter, to remove the aborting cows from the byre. One must then at least place them in a separate row, and take care that the discharge from their genital organs is not directly through the channel for the urine, or indirectly through the byre utensils, brought into contact with the vaginal orifice of healthy pregnant cows. In such circumstances it is also well to carry out prophylatic disinfection, such as washing of the external genital parts and their neighbourhood

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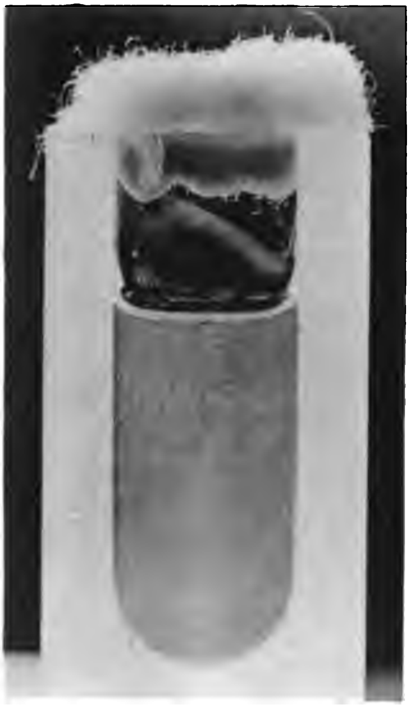
[1] It is interesting that five of these cows aborted at ten weeks, one at three months, and one at four-and-a-half months, before the normal end of pregnancy. This shows that the abortion catarrh may have an insidious course even when the bacilli are introduced through the act of copulation. The observation is well calculated to strengthen belief in the importance of the bull as an intermediary bearer.



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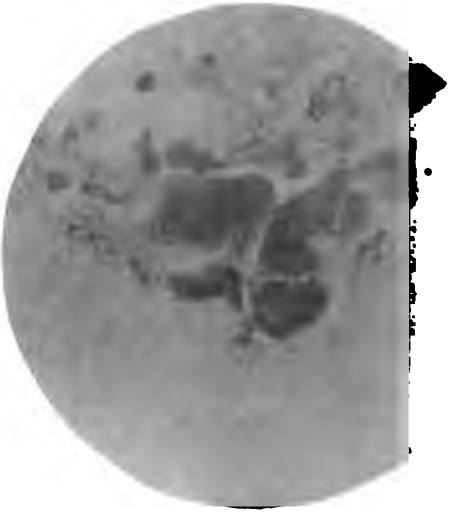

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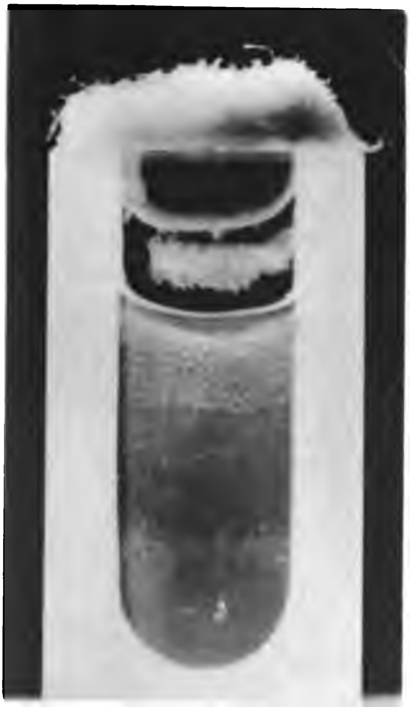

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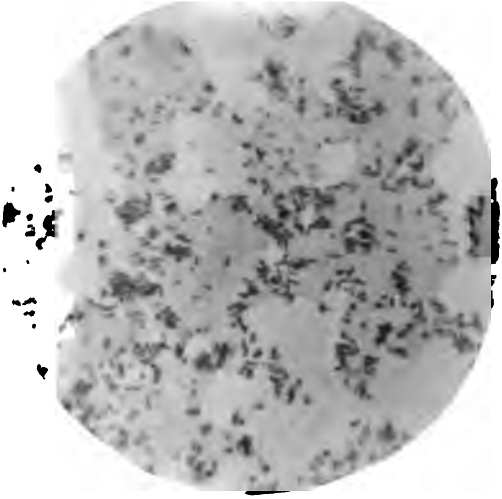

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in the pregnant animals, and perhaps also to practise prophylactic vaginal injections.

Careful cleansing and disinfection of the floor of the byre are also to be recommended. Under the head of disinfecting agents I would call especial attention to the free use of powdered lime; that is sometimes used by the farmers in this country, and it has the advantage of not imparting any disagreeable smell to the milk, and of not diminishing the value of the dung.

The uterus of the cow that has aborted must be submitted to a careful disinfectant treatment, and the cow must not be put to the bull until she appears to be quite clean; even when this is strictly attended to the same bull ought not to be used for healthy cows. If this cannot be carried out, the preputial cavity of the bull must be washed out with a disinfectant solution every time after he has served a suspected cow.

As has been specially insisted upon by Sand, the system recommended by the older authors of selling the cows that have aborted, and replacing them by newly purchased animals, is not a good one. A careful disinfection of the uterus will, in many cases, succeed in freeing the cows from the specific uterine inflammation, so that they will carry to full term at next pregnancy; and even when this result is not obtained it is certainly better to bear the loss caused by repetition of the abortion, in expectation of immunity setting in, than to provide fresh fuel for the fire. One must certainly admit that this method may be associated with great loss, but here, as is often the case, the first loss is probably the least.

Inasmuch as epizootic abortion belongs to the diseases which leave behind them at least a relative immunity, the possibility of conferring such immunity by prophylactic injections of a vaccin or of an abortion serum naturally suggests itself. To the future must be left the task of accurately investigating this question as well as that of the possibility of a serum therapeutics. Perhaps nothing can be done in this way. Fortunately, however, it will be possible to successfully combat the plague by circumspect use of the old approved means — isolation and disinfection.

EXPLANATION OF PLATE II.

Fig. 1. Colonies of abortion bacilli in a tube of agar-serum. Natural size. (The spots above and below the zone of growth are due to cracks in the glass.)

Fig. 2. Colonies of abortion bacilli in a tube of agar-serum which was submitted to a pressure of five atmospheres. Both optima-zones are shown, and also a few colonies in the intermediate zone. Natural size (in this figure also there are some spots due to cracks in the glass).

Fig. 3. Uterine exudate with large cells completely filled with bacilli. Many free-lying bacilli can be seen (but very indistinctly) by the aid of a lens. The preparation is contaminated with some large bacilli. Magnified 500.

Fig. 4. Abortion bacilli from a bouillon-serum culture, stained with fuchsin. Magnification 1000.

For the photographs I am indebted to the kindness of Professor V. Storch.

THE EXAMINATION OF COW'S MILK FOR THE DETECTION OF PATHOGENIC PROPERTIES.

By SHERIDAN DELÉPINE, M.B., B.Sc., Procter Professor of Pathology, Owens College, Victoria University.

THE examination of milk as generally conducted for public health purposes has for chief objects the detection of impurities or alterations indicating clearly fraudulent admixtures or absence of the necessary care to prevent gross contaminations.

Comparatively simple, physical, and chemical methods have, with these objects, been used by public analysts.

After taking the reaction, the specific gravity, the amount of fat, and sometimes the total amount of solids other than fat, the chemist can generally tell whether water has been added, cream abstracted, or unfavourable fermentations have taken place. He may, by further examination, be able to recognise the presence of such substances as carbonate or chloride of sodium, carbonate of calcium, salicylic acid, boracic acid, glycerine, gum arabic, starch, etc., etc., when these have been added for some purpose or other.

By a microscopical examination some useful information may also be gained. Cells or granules, such as starch corpuscles, hairs, fungi, débris of food or litter, blood, pus corpuscles, colostrum corpuscles, etc., may easily be detected.

Such examinations make it possible to exclude from the market milk which does not come up to certain standards of purity; exclusion which from the sanitary point of view, and quite apart from the legal aspect of the question, is very desirable. There can be no doubt that milk which has been adulterated or grossly contaminated has not only inferior feeding qualities, but is liable to produce disease. It is therefore unnecessary to insist upon the importance of ordinary methods of milk inspection and analysis.

It may be well, however, to consider whether such methods are sufficient to establish the absence of noxious properties. In other words, being admitted that when milk is shown by ordinary analysis to be of bad quality it should be condemned, is it certain that milk which is not thus condemned will be free from bad properties?

Numerous outbreaks of disease attributed to milk have been recorded during the last quarter of a century, and these seem to prove that milk is an important factor in the dissemination of disease, and that it is not easy by the methods of inspection at present in use to protect the public against this danger.

Mr Ernest Hart has published in 1881 and 1897 two series of articles on milk-borne disease, which show clearly the extent of the danger, and the interest taken in this question is well shown by the notoriety acquired by Power and Klein's investigations in connection with the Hendon outbreak.

Among the diseases said to be communicable by milk, it is enough to mention tuberculosis, diphtheria, scarlet fever, typhoid fever, cholera, aphthous stomatitis, various forms of gastro-enteritis, giving rise to diarrhœa, vomiting, and various other morbid manifestations. All these diseases are liable to occur in our country. It is unnecessary here to allude to forms of intoxications or infectious diseases which may occur elsewhere.

It would be a matter of great importance to discover how milk acquires the noxious properties which give rise to so much mischief The cow has been blamed in a great number of cases, and on good grounds. If disease of the cow were the only cause of contamination of milk, it is evident that careful inspection of stables by competent veterinary inspectors should do much to stop the evil, and what has already been done in this direction has given, so far, results which justify our expectations.

But milk is exposed to many contaminations after milking, and it is important to discover also the share which these contaminations have in the production of disease.

For many years I wished to study this question, but as such a study requires the command of a large amount of material, and access to many sources of milk supply, I was able only to make now and again a few occasional observations. In the course of last year I was, however, suddenly supplied with the material I wanted.

Both in Manchester and Liverpool the Medical Officer of Health independently determined to enquire how far the milk supply of those towns was to blame for the spread of tuberculosis.

The object of the Medical Officers of Health of Liverpool and Manchester, was therefore to ascertain how much of the milk supplied in these towns was tuberculous.

Dr Hope dealt with the usual milk supply of cities so as to obtain a measure of the risks of infection from the mixed milk usually found on the market. He had altogether 167 samples of milk examined by Professor Hamilton, Professor Boyce, Dr Woodhead and myself, and the results of these examinations will be found in Dr W. E. Hope's report.[1]

In this communication I will confine my remarks to the specimens I have examined myself.

In Manchester the investigation was conducted in a different manner, and formed part, so to speak, of a sanitary inspection of the cowsheds. The chief object of the examination of milk was to discover in what proportion of the cows found in a state of advanced tuberculosis the milk was capable of communicating tuberculosis. The result of this very important investigation will be found in Dr Niven's report.[2]

Before and since the beginning of these two enquiries I have also made a number of independent observations on milk infections of various kinds. Putting aside a number of examinations the results of which have yet to be submitted to Public Health Departments. I have as a basis for discussion, the results of some seventy examinations of milk, viz.:—

Thirty-six specimens of mixed milk from Liverpool, sent by Dr Hope.

Twenty-four specimens of unmixed milk from Manchester, sent by Dr Niven.

Ten specimens of unmixed milk from Manchester, collected by myself.

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[1] Report of the Medical Officer of Health on Tuberculosis as affecting the milk supply of the city, Liverpool, 6th May 1897.

[2] Report on the Manchester Cowsheds, etc., Manchester, 1897.

It will be convenient to consider separately the results obtained with the thirty-six mixed milks, and afterwards those found in the case of the thirty-four unmixed milks. Having already summed up in a report to Dr Hope the results which I have obtained with the mixed milks which he sent me from Liverpool, this report may with advantage be reproduced here.

I am much indebted to Dr Hope for kindly supplying me with information as to the sources from which the samples I had examined from him were derived. This information was given to me only after the issue of my report.

REPORT ON SPECIMENS OF MILK SENT BY THE LIVERPOOL MEDICAL OFFICER OF HEALTH FOR BACTERIOLOGICAL EXAMINATION.

4th April 1897

1. In submitting this report on specimens of milk which have been sent for investigation to me, by Dr Hope, I wish to explain briefly the methods which I have used.

In investigations of this kind the value of the report depends entirely on the methods used and on the care with which they have been carried out.

Too much care cannot be expended on researches the results of which may serve on the one hand to reveal a public danger, and, on the other hand, cause serious losses to certain members of the community.

To find whether a certain sample of milk is capable of causing tuberculosis two methods can be followed. (1st) The parasite causing the disease (the bacillus tuberculosis) may be looked for with the microscope in the milk; (2nd) The effect which this milk has on animals may be tested by direct experiments.

Young animals susceptible to tuberculosis may be fed on the milk in question, or small quantities of the same milk may be injected under the skin or into the peritoneum. When tubercle bacilli are present in the milk *in sufficient numbers to be a source of danger*, tuberculosis will be produced in the inoculated animal. As I have shown several years ago,[1] by inoculating guinea-pigs with very small quantities of tuberculous products under the skin of the hind leg, on the inner aspect of the thigh, distinct tuberculous lesions are produced within twelve days, and these become more and more distinct during the following week. Such lesions bear quite distinct relations to the seat of inoculation, so that it is possible to clearly recognise the lesions which are the result of the inoculation from those which might have been produced accidentally in some other way. The adjoining diagrams show the effects of subcutaneous inoculation of tuberculous matter of ordinary virulence (*e.g.*, tuberculous sputum) at various intervals after inoculation on the inner aspect of the leg at the level of the femoro-tibial articulation. They correspond to four stages which I have adopted for descriptive purposes: I. 1st stage, There is a local lesion at the seat of inoculation. At that date the popliteal, inguinal, and even the sublumbar ganglia are usually also swollen

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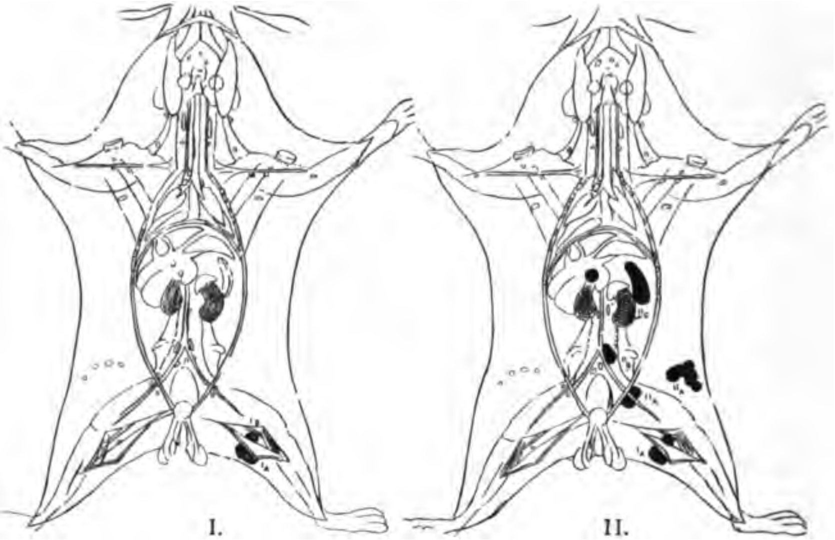
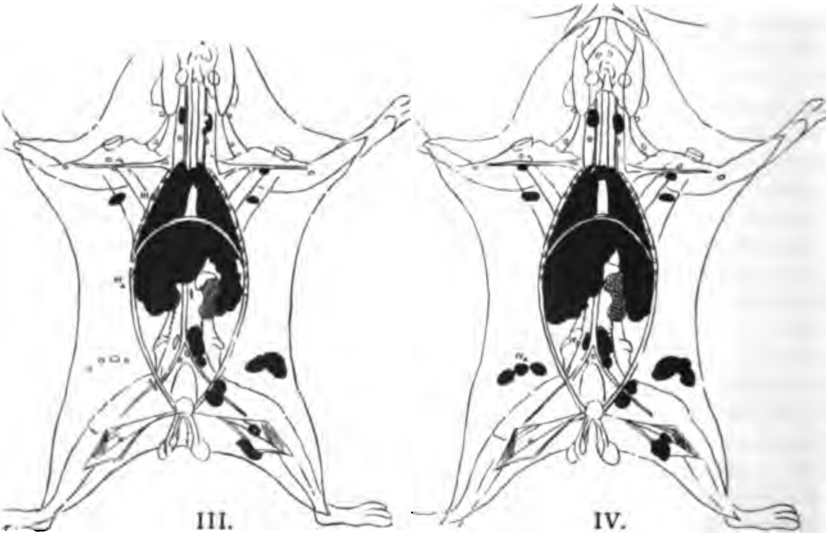
[1] **On the Value of Experimental Tuberculosis in Diagnosis. B. M. J., Sept. 1893, and several subsequent papers in the British Medical Journal and the Medical Chronicle, Manchester.**

DIAGRAMS OF TUBERCULOUS LESIONS IN GUINEA-PIGS INOCULATED ON THE INNER ASPECT OF THE LEFT KNEE WITH VERY MINUTE QUANTITIES OF TUBERCULOUS MATTER.



Less than 10 days after inoculation. *10 to 15 days after inoculation.*



20 to 30 days after inoculation. *50 days or more after inoculation.*

and congested, and the first two may also show patches of necrosis. II. 2nd stage, All the lymphatic ganglia between the seat of the lesion and the diaphragm on the side inoculated are distinctly affected; the retrohepatic ganglion and the spleen are also involved. III. 3rd stage, The same organs as above are more diseased, and, in addition, the liver, the bronchial ganglia, and the lungs are affected, and several other lymphatic ganglia, *on both sides* of the body, above the level of the diaphragm. IV. 4th stage, Same organs as above, more diseased. In addition to them the lymphatic ganglia of the side *not inoculated*, and below the level of the diaphragm are beginning to be involved. In all these diagrams I have drawn the tuberculous lesions black. These diagrams indicate what can be recognised by means of a naked-eye examination only.

I have also demonstrated (by a series of several hundred experiments) that when proper precautions are taken (*isolation of animals, disinfection of cages, good ventilation of animal house, clean feeding, inoculation under strict aseptic precautions*) the most varied pathological products (other than tuberculous) can be introduced under the skin of the guinea-pig without producing lesions that can be mistaken for tuberculosis.

Many observers are still under the impression that intraperitoneal inoculation is preferable to subcutaneous inoculation; this is undoubtedly an error. The peritoneal cavity is not more sensitive to tuberculous infection than the subcutaneous tissue, the chances of accidents from other infections are greater, the relation of the lesions to the inoculations are not so clearly established, and by proper devices it is possible to introduce under the skin as large an amount of material as is necessary for the purposes of the test.

2. From the above it will be seen that I have special confidence in subcutaneous inoculation of guinea-pigs as a test for tuberculosis. This, however, has not prevented me from using other methods, for I thought they might furnish important additional information.

In order that the examination of the specimens should be as useful as possible, I have thought it also desirable in each case to apply a few tests showing the state in which the specimen was when I received it and, roughly, the quality of the milk. For this purpose I have, whenever possible, taken the reaction, the specific gravity, the amount of cream that could be separated in a quarter of an hour by means of the centrifugal machine (giving 3000 revolutions to the minute), the amount of sediment, the number of cells, and the total amount of bacteria present.

In all cases the specimens were examined the same day as I received them.

3. Even if tubercle bacilli were equally distributed through tuberculous milk (which they are not), a very large number of them would have to be present before one could expect to find one bacillus now and again in the few drops of milk used for microscopical examination.

To examine completely 1 ounce of milk microscopically it would be necessary to make at least 500 microscopical examinations, for not more than one drop can be examined at a time. If there were about 100 bacilli in that ounce of milk it would be necessary to examine on an average five drops of it before one bacillus was discovered. To prepare and examine each drop thoroughly does not

The animals were then kept from the very beginning in separate disinfected cages in a warm, well-ventilated and well-lighted animal house. Under these conditions, in most cases, they seemed to suffer in no way from the injection, and a few minutes after the injection they looked as well as before. In a few cases, however, the milk proved highly virulent and killed the animals within forty-eight hours. Such milk when taken uncooked in large quantities would probably cause gastro-intestinal troubles. With the exception of the few that died thus, and of some others that died from accidental causes unconnected with the inoculations, all the other animals were kept three, four, and sometimes up to seven weeks. They were then killed and a careful *post-mortem* examination made.

5. In the case of the animals inoculated in the leg all the lymphatic glands situated on the lymphatic paths carrying the lymph from the inoculated part were examined; so were the spleen and the liver and very often other viscera as well.

In the case of animals inoculated in the peritoneum, the mesenteric ganglia, the spleen, the liver, the diaphragmatic and pelvic lymphatics and the peritoneum itself were minutely examined.

Tuberculous lesions of some of these parts can be recognised easily with the naked eye from ten days to two weeks after inoculation, and after four weeks they are in all quite unmistakable. Yet to leave no room for doubt, whenever the lymphatic ganglia, the spleen, or the liver showed any signs of inflammation they were examined microscopically with a view to discover whether tubercle bacilli might be present against all expectations. In some cases I have also isolated the bacteria that had caused acute inflammatory lesions of the peritoneum, subcutaneous tissue, lymphatic ganglia, or spleen.

A bacillus resembling closely, if not identical with, the bacillus coli communis has been found to be the cause of rapidly fatal peritonitis in several cases, and seems to have been the cause of death in at least one more case. In some other cases cocci and other micro-organisms have been found in the lesions.

6. All the inoculations and the *post-mortem* and bacteriological observations recorded in the tables appended in this report have been made by myself. Owing to the large accumulation of material which had to be examined microscopically I have been obliged to obtain some help for the staining and examination of many microscopical preparations, and I have to thank Dr F. C. Moore for the very able assistance he has given me in this respect. To Dr E. J. Sidebotham I am much indebted for the care with which he has submitted the various organisms I isolated from the lesions produced by the milk injections to numerous cultural tests. To obtain perfect uniformity in the results, however, I have examined myself all the preparations without exception, so that I am personally responsible for all the statements which are made in this report as well as for the methods used.

7. As an additional precaution to eliminate several sources of error, I have indicated in the table the number of hours which have elapsed between the time when the cows were milked and the time when I made the inoculations and examined the specimens. I have also separated from each other various lots of milk received at different periods, so that if there was anything in the way in which they

had been collected that had caused them to become contaminated this would be at once apparent. I think there is only one lot in which the suspicion of such an accidental contamination of the sample might be entertained, and that is the last (specimens 400, 401, 402); yet in this group the milk had been kept a shorter time than in almost any other lot, and showed no special evidence of gross contamination.

(To be continued.)

THE ETIOLOGY OF PNEUMONIA IN THE PIG.

By J. M'FADYEAN, Royal Veterinary College, London.

(Continued from page 13).

CASE NO. VI.

THE specimens received in this case were the lungs, heart, cæcum, and part of the colon of a pig. The pleura over the right lung was almost everywhere covered by a thin layer of greyish fibrinous lymph, and nearly three-fourths of the organ was in a condition of firm hepatisation. The condition of the left lung was similar, but the pleurisy was less extensive. The cæcum and colon showed swine-fever ulceration.

A cover-glass preparation from the hepatised lung tissue, stained with methylene blue, showed only a few short bacteria.

A particle of lung was dug with sterile instruments from the depth of the hepatised lung tissue and shaken up with sterile water, and of the turbid liquid thus obtained 1 cc. was injected under the skin of the abdomen of a rabbit.

The rabbit was found dead nineteen hours after inoculation. The seat of inoculation showed some inflammatory œdema, and a cover-glass preparation from the ear blood showed an enormous number of short bacteria of the fowl-cholera type. Three agar and two bouillon tubes were inoculated from the blood in the heart. The agar tubes, after twenty-four hours' incubation at 37° C., yielded an abundant growth, the discrete colonies being flat, circular, and translucent. The bouillon tubes at the same time were slightly turbid, but with continued incubation the liquid soon became clear and deposited a rather coarse flocculent precipitate. All these cultures were found to be composed of short or ovoid bacteria, not distinguishable morphologically from those of fowl cholera.

No experiments on pigs have been made with this organism, but its cultures have been proved to be very virulent for the rabbit, death occurring within twenty-four hours after subcutaneous inoculation with moderate quantities. In gelatine and gelatine-agar it forms spherical or oval white opaque colonies, but no appreciable growth takes place on potato.

Finally, as an appendix to the account of this case, it may be stated that the lungs of a second pig of the same lot were received along with the above-described organs. The lungs showed lesions similar to those in the first pig, and a cover-glass preparation from them showed numerous short or ovoid bacteria. A rabbit inoculated, with the usual precautions, from the hepatised lung tissue was found dead twenty-eight and a half hours afterwards, and an organism apparently identical with that recovered from the first pig was abundantly present in the blood, and grew with similar characters in artificial media.

CASE No. VII.

The parts received in this instance were the lungs, heart, and cæcum of a pig, unaccompanied by any history. The heart was covered by a thin layer of fibrinous lymph, easily detachable in the form of coherent shreds. The right lung, opposite the heart, was the seat of an area of hepatisation about the size of one's fist. This area on section showed a coarsely mottled, dark red, and yellowish surface. The cæcum appeared healthy.

The parts when received were far from fresh and no bacteriological examination of them was made, but the following experiments were carried out in order to test the transmissibility of the disease.

The hepatised area was separated from the rest of the lung, and immersed for half a minute in boiling water. The object of this was to destroy any swine-fever bacilli that might accidentally be present on the exterior of the lung.[1] With a sterile knife an incision was then made into the solid part, and the cut surface was scraped with the knife. A quantity of the material thus obtained was mixed with 1 cc. of sterile water and injected under the skin of a pig in the region of the sternum. The hepatised part was then cut into small pieces with a sterile knife and mixed with the food of five pigs, viz., the before-mentioned inoculated pig and four others. The pigs were about ten weeks old, and they were all kept together.

The whole of the five pigs were killed on the eleventh day of the experiment. In the interval they had not shown any symptoms of disease, save that in the inoculated pig a walnut-sized lump had formed beneath the skin at the point where the liquid had been injected. At the *post-mortem* examination this swelling was found to be an abscess. The abdominal and thoracic viscera of all the pigs appeared to be normal.

CASE No. VIII.

The parts sent for examination in this case comprised a piece of large intestine with characteristic ulcers, and three pieces of lung from the same pig. Two of the pieces were each about the size of the two fists, and fully the half of each was solid from hepatisation; the third piece, which was somewhat smaller, was two-thirds hepatised. On section the hepatised portions showed slight swelling of the interlobular septa. The colour of the cut surface was in some places dark red, and suggestive of infarction or hæmorrhage; elsewhere the surface had a mottled appearance—greyish and light red. The bronchi in the hepatised parts contained muco-purulent matter. A cover-glass preparation from the hepatised part showed immense numbers of short bacteria, while similar preparations made from the spongy lung tissue showed only a few variously shaped organisms (lung not quite fresh).

A rabbit was inoculated subcutaneously in the right flank with 1 cc. of sterile water holding in suspension a scraping from the depth of one of the hepatised areas in the above lung. No obvious effect was produced by this inoculation, and the rabbit was alive and apparently well five weeks after inoculation.

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[1] The specimens had been sent from the offices of the Board of Agriculture, and there was therefore reason to suspect that they might have been in contact with swine-fever organs.

Three slanting agar tubes inoculated from the depth of one of the hepatised areas in the above lung yielded, after twenty-four hours' incubation at 37° C., an abundant culture, in one of the tubes continuous, and in other two composed of discrete colonies; at the end of twenty-four hours these discrete colonies had attained a diameter of 3 mm. to 4 mm. The colonies were approximately circular in shape, slightly raised in the centre, glistening, and opaque white. On microscopic examination they were found to be composed of very short, almost regularly ovoid bacteria. When cultivated in boullion this organism was found to grow in the form of coarse flocculi, the intervening liquid remaining nearly transparent. It is non-motile, forms a meagre white growth on potato, and coagulates milk.

The three hepatised areas of lung referred to above were separated from the still spongy lung tissue, and then immersed for half a minute in water at the boiling point. The pieces of lung had been sent wrapped up together with the piece of ulcerated swine-fever bowel, and the object of this immersion in boiling water was to destroy any swine-fever bacteria that might accidentally have been deposited on their surfaces. Each of the three pieces was about as large as the fist, and the half-minute's immersion in boiling water had only coagulated a thin stratum of lung at the surface of each. All three pieces were consumed by an adult sow.

The sow was killed on the eleventh day of the experiment. In the interval it had not shown any symptom of ill-health, and at the *post-mortem* no lesions were discovered in any of the viscera of the abdomen or thorax. No further experiments have been made with the organism isolated from this case.

CASE NO. IX.

The parts received in this case comprised pieces of large intestine and several pieces of lung from a pig. The pig had been found dead in the morning of the day on which the parts reached the laboratory. The pieces of large intestine showed unmistakable lesions of swine fever. In the pieces of lung there were several sharply defined very firm areas, the largest being as big as a goose egg. On section these solid areas showed the appearance of necrotic lung tissue, the cut surface being dry, firm, and yellowish-white in colour.

The surface of one of these solid areas was seared with a red-hot spatula, and by means of sterile instruments a small piece was dug out from the centre of the part and used to inoculate the surface of slanting agar in test tubes. The tubes were afterwards placed in the incubator at 37° C., and on the following day there had formed in each an abundant growth, for the most part continuous, but at some places composed of discrete colonies. These colonies were flat, and circular in outline, and both they and the continuous growths were very translucent. From the growths in one of the tubes plate cultures were made with gelatine-agar, and these yielded a perfectly characteristic pure growth of swine-fever bacilli.

A pig about two months old was fed with 100 cc. of a bouillon culture, which was the fourth generation from a discrete colony in one of the gelatine-agar plates. The culture had been incubated for forty-eight hours at 37° C., and it was uniformly turbid, but without any surface growth or scum. A tube of gelatine agar inoculated from the flask subsequently yielded a characteristic growth of swine-fever bacilli along the needle track.

On the second day of the experiment the pig was dull, huddled up in the straw, and taking little or no food. It was passing an abundance of thin greyish fæces. On the third and fourth day the general condition of the pig was much the same, but the diarrhœa was more profuse. On the fifth day it appeared to be more lively, and took a little food.

The pig was killed on the sixth day of the experiment. The *post-mortem* examination showed inflammatory congestion of the mucous membrane of the cæcum and colon, with about half-a-dozen small ulcers from which the slough had already separated. Two slanting agar tubes inoculated from a mesenteric gland yielded numerous colonies of swine-fever bacilli, which, when re-inoculated as stab cultures, grew with the characteristic form in gelatine-agar.

CASE NO. X.

The parts received in this case were the heart, lungs, and a piece of the large intestine of a pig. The latter showed characteristic swine-fever ulcers. The pericardium was everywhere adherent to the heart, as the result of a dry pericarditis. The adhesion was so firm that the pericardium could with difficulty be stripped off the heart. The surface of both lungs showed evidence of pleurisy in the shape of a layer of fibrinous lymph, which was most abundant on the base or diaphragmatic surface of each lung. The mediastinal lymphatic gland was enlarged to about half the size of the little finger, and on section it was firm, with a mottled greyish and yellowish-white colour. The bronchial and tracheal lymphatic glands were in a similar condition, one of the latter being as large as a garden bean.

From one of the enlarged lymphatic glands a piece was dug out with heated instruments, and rubbed over the surface of two slanting agar tubes. The tubes were placed in the incubator at 37° C., and on the following day each of them showed a continuous translucent growth. From the growth in one of the tubes a stab culture was made on gelatine-agar, and this grew with the characteristic appearance of stab cultures of the swine-fever bacillus in that medium.

CONCLUSIONS.

It will be recollected that one of the main objects of these experiments was to ascertain the cause or causes of pneumonia in pigs, and more particularly to test the correctness of the still widely held opinion that a genuine swine-fever pneumonia is of frequent occurrence. The general result of the experiments will be gathered from the following summary. In seven of the cases, viz., Nos. I., II., IV., VI., VIII., IX., and X., the pig had been the subject of swine fever, as proved by the presence of characteristic lesions in a portion of large intestine forwarded along with the lungs. In one other case (No. III.), although I was unable to recognise characteristic lesions in the accompanying piece of bowel, it may be taken as probable that the animal was affected with swine fever, since that disease had been diagnosed by the experienced practitioner who sent the specimens. In the two remaining cases (Nos. V. and VII.) there was no evidence of swine fever in the bowel. In only two of the cases (Nos. IX. and X.) did the experimental and bacteriological examination indicate that the swine-fever bacillus was present in the thoracic organs. Strictly speaking, the last of these two cases has no bearing on the main point of the present inquiry, for while the pig had been the subject of pleurisy and pericarditis, the lungs were not the seat of pneumonic lesions. The case is, nevertheless, interesting in the present connection, as showing the ease with which the swine-fever bacillus can be detected when it is present in comparatively fresh organs.

The net outcome of the investigation therefore is that in only one out of nine cases of pneumonia (seven of which had at the same time swine-fever lesions in the bowel), was the pulmonary lesion ascribable to the swine-fever bacillus. In this one case (No. IX.) the lung lesions were of a quite exceptional character, and took the form of well-defined firm dry areas of necrosis. It has never been my lot to encounter an identical condition of lung in a natural case of swine fever, but they closely resembled the lesion which developed in an unpublished experimental case, in which the pig was infected by the injection of a bouillon culture of the swine-fever bacillus through the chest wall into the lung tissue.

It must therefore be regarded as certain that there is a genuine swine-fever lung lesion, but that this lesion so rarely occurs in natural cases of the disease as to be almost devoid of practical interest. The admission that the swine-fever bacillus does sometimes determine pulmonary lesions cannot be allowed to affect the only safe rule for guidance in the diagnosis of swine fever, viz., that the opinion must be based on the condition of the large intestine, which in this disease is never normal, and is in all save very acute cases the seat of lesions that are quite characteristic.

While the main point of the inquiry may thus be considered settled, a question of some importance still remains to be answered, viz., if the swine-fever bacillus was the cause of the pulmonary lesions in only one of the nine cases (that is excluding Case X., in which only pleurisy was present), what was the cause in the other cases? The answer to this question is, that the organisms isolated from the hepatised lung in the several cases were probably accountable for the lesions. In six of these cases (the first six of the series) the organism isolated was a short or ovoid bacterium, whose morphological characters are sufficiently described by saying that it was not distinguishable by shape or size from the fowl-cholera bacterium. Furthermore, the organisms isolated from these six cases closely resembled the fowl-cholera germ in staining reactions and mode of growth in the common artificial media. A perusal of the record of the experiments will show that in spite of their morphological identity the organisms isolated from these six cases varied considerably as regards their effects when inoculated into rabbits and pigeons, but one would not be justified in inferring from these differences that they represent distinct species.

It need hardly be said that in describing the organisms as morphologically identical with the fowl-cholera bacterium it is not intended to suggest that there is any etiological connection between pneumonia of the pig and fowl cholera. The fowl-cholera bacterium was the first described of a series of organisms easily enough distinguished from one another by their effects on different animals, though closely alike or identical as regards their morphological, staining, and cultural characters. To this group belongs also the organism which Loeffler and Schütz isolated from diseased pigs in Germany, and which these authors named the Schweinseuche (swine plague) bacterium. It follows therefore that the organisms isolated from Cases I. to VI. in the preceding series of experiments are morphologically identical with the bacterium which is generally accepted as the cause of an infectious pneumonia of pigs in Germany, and they do not present any character by which I can distinguish them from that organism. I am very far, however, from concluding from the previously described experiments that we have in this country a second epizootic disease of the pig in the form of an infectious pneumonia.

Want of space makes it impossible to discuss this possibility at length on the present occasion, but it may be pointed out that the various inoculation and feeding experiments made with pigs had not the results that one would have expected on the assumption that the bacteria employed were the cause of disease of an epizootic character. Besides, on clinical grounds it is well nigh impossible to believe that a pneumonia of the pig with markedly infectious tendencies can exist in this country, and yet have escaped recognition. It therefore appears probable that the organisms which are generally accountable for pneumonia in British pigs belong to the class of facultative parasites, which as a rule lead a saprophytic existence, and only occasionally cause disease by propagating in the living body. It is even conceivable that these organisms may frequently inhabit the alimentary canal or air passages of healthy pigs, and only become pathogenic when, owing to some sudden access of virulence on their part, or some state of diminished resistance on the part of the pig, they are able to invade and propagate in the living tissues. But while it is thus probable that most cases of pneumonia in the pig in this country are sporadic, it is perhaps best to keep an open mind on the subject ; and it is to be hoped that when those who are in practice encounter what appear to be outbreaks of pneumonia in the pig they will take the trouble to enquire closely into the previous history of the animal, and place on record any facts which either indicate or contra-indicate contagion as the source of the disease.

EDITORIAL ARTICLES.

SWINE ERYSIPELAS.

AT a later part of this number there will be found the clinical history of a series of cases of swine erysipelas which were traced by Mr Hedley, of the Veterinary Department of the Irish Privy Council. The history of these cases was traced as part of an investigation conducted by the Departmental Committee which was appointed by the Board of Agriculture in 1895 to inquire into the etiology, pathology, and morbid anatomy of the diseases classed as swine fever. The report is of great interest and value, inasmuch as it furnishes precise information regarding a subject that has hitherto been surrounded by a good deal of obscurity.

Since 1891, when a number of cases of the disease were put on record in this Journal,[1] it has been known that pigs in this country are not exempt from the disease which is termed *Schweine Rothlauf* in Germany and *rouget du porc* in France.

Prior to the date mentioned this disease had not been recognised as a common affection in Great Britain or Ireland, and indeed it was not known that it ever occurred among our pigs. The disease had long been known as a formidable plague of the porcine species in France, Germany, and other Continental States; and as soon as its existence here was proved, the possibility that it was of common occurrence, but overlooked owing to its being confounded with swine fever, naturally suggested itself. As a matter of fact it appeared highly probable that if it existed here at all it could not be a rare disease, since on the Continent it was regarded everywhere as a highly contagious and infectious malady.

In the cases which were first detected in England the lesions took the form of endocarditis with luxuriant vegetations on the cardiac valves, and this is probably the explanation of the fact that even at the present time many veterinary surgeons in this country believe that cardiac lesions are constant in swine erysipelas, and do not recognise the disease by any other mark. That, however, is an erroneous view of the disease, if reliance may be placed on the account of it which is given by all foreign authors. According to these accounts swine erysipelas is in most cases an acute septicæmic disease of a highly fatal character. The duration of the illness usually extends to only a few days, and the mortality is often as high as 50 per cent. In these rapidly fatal cases the only lesions usually encountered at the *post-mortem* examination are an erysipelatous inflammation of the skin, swelling of the spleen, and gastro-enteritis without ulcerative or diphtheritic changes in the mucous membrane.

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[1] Vol. IV., p. 316.

Animals that do not succumb within a few days generally make a prompt and apparently complete recovery, but in occasional cases the pig remains unthrifty, develops symptoms of heart disease, and dies suddenly after some weeks or months.

These accounts of the disease naturally suggested the thought that the cases of swine erysipelas with heart lesions occasionally detected in this country did not by any means represent the real extent to which the disease prevailed among British pigs, but that pigs so affected were merely a few of the survivors from outbreaks in which most of the animals had died from the acute form of the disease. This possibility was suggested in the article to which reference has already been made, although, as was then pointed out, the history of the cases recorded did not indicate that the disease was of a highly infectious character.

When swine fever was taken in hand by the Board of Agriculture the *post-mortem* examination conducted by the officers of the Board soon showed that at least the chronic form of swine erysipelas was by no means rare, for many cases with cardiac lesions were discovered among pigs that had died from swine fever or been killed as suspected of that disease. The operations of the Board, however, did not suffice to clear up the point of greatest interest and importance, since no attempt was made to trace the previous history of these chronic cases in order to find out whether other animals of the same lot had suffered from the acute rapidly fatal form of the disease, or whether there was any evidence that the disease was spreading by contagion.

It was to fill up this blank in our knowledge of the disease as it occurs in this country that the investigations recorded in Mr Hedley's report were undertaken. A study of the information given in that report makes it evident that the accounts of swine erysipelas which are given in foreign text-books do not exactly apply to the disease as it occurs with us. If swine erysipelas is a disease that is readily spread by direct or indirect infection—and such is the view of it generally current abroad — one would expect to find it attacking the whole or a considerable number of the pigs kept on the premises where it has obtained a footing ; and when the past history of each case of the disease is obtainable, one would also expect to be able to trace a connection with an antecedent case. This, however, is not the state of affairs disclosed in Mr Hedley's series of cases. Take for example Case R. In this case the owner of the two pigs attacked with the disease had not previously kept animals of that species, and these two pigs had been two months on his premises before the disease showed itself. The two pigs were seized with the acute form of the illness nine weeks after purchase, and one of them promptly died, while the other subsequently developed the symptoms indicative of heart affection, and was found to have vegetations on the cardiac valves when killed six weeks after the onset of the disease.

In this case the disease must have been contracted on the premises where the pigs were attacked, for nine weeks is an impossible period of incubation in swine erysipelas. Besides, four other pigs that had been in contact with the two subsequently attacked remained healthy.

Again, take Case S. In this case only two pigs out of a total of twenty on the premises appear to have been attacked. The two affected pigs had been bred on the premises, and three of the same lot remained healthy. There had been no new purchases for nearly two months, and the pigs last bought had never shown any symptom of illness.

Case U. is equally opposed to the idea of infection from a previously diseased pig. The owner had only two pigs on his premises, and they had been in his possession for over eight months before the disease showed itself. During that time the two pigs had not been in contact with any others. One died from the disease, and the other escaped.

The whole series of cases makes it impossible to believe that the disease is markedly contagious, like swine fever for example. The impression which the history of the cases leaves on one's mind is that swine erysipelas and swine fever belong to two perfectly distinct categories of disease. The latter is a strictly contagious and infectious malady, caused by a parasite which, in natural circumstances, probably has the body of the pig as its exclusive field of propagation—in other words, every case of swine fever has for its starting-point the ingestion (or admission into the body by some other channel) of bacteria that were bred in the body of an antecedent case of the disease. Swine erysipelas, on the other hand, appears to be usually a sporadic disease, caused by a germ which is only a facultative parasite—that is to say, which frequently or generally propagates outside the body of the pig, and is only occasionally the cause of disease.

The distinction is of great practical importance. If there were good reasons for supposing that swine erysipelas is a disease mainly spread by contagion, it would have to be combated by just the same measures as are considered necessary for the suppression of swine fever. If on, the other hand, the disease is caused by a germ that has a wide distribution in nature, like, for example, the pyogenic organisms or the tetanus bacillus, it would be transparently foolish to endeavour to suppress it by the stamping-out measures now being enforced against swine fever.

It does not follow, however, that because swine erysipelas is usually sporadic, and therefore not to be combated by such summary and expensive measures as wholesale slaughter of diseased and suspected, it can be altogether ignored from a prophylactic point of view. As far as can be at present seen there is a certain analogy between swine erysipelas and anthrax as regards the origin of outbreaks. In both cases contagion and infection, in the ordinary sense of these words, have little to do with the origin of cases. The germs of anthrax at the present moment exist in the soil in many places, and perhaps even propagate there, and it is the accidental ingestion of such germs that determines most outbreaks. Very probably cases of swine erysipelas have a similar etiology. But although we do not slaughter animals because they have been in contact with one affected with anthrax, we recognise the necessity of taking steps to prevent further ground contamination, and prudence directs that we should do the same whenever a pig dies from swine erysipelas. A pig dead of that disease contains countless numbers of the specific bacilli, and these are probably also voided in its excretions. These considerations, therefore, suggest such precautionary measures as disinfection and safe disposal of the carcase.

Fortunately, however, there appears to be a great difference between anthrax and swine erysipelas in regard to the virulence of their respective microbes, and the facility with which animals may be infected by them. We can with great certainty infect animals belonging to susceptible species by feeding or inoculating them with material containing anthrax germs, but the experiments carried out for the Departmental Committee indicate that swine erysipelas is not very readily transmitted, at least to British pigs, by either of these methods. In these experiments attempts were made to infect pigs of various ages with swine erysipelas, but the attempts were uniformly unsuccessful, whether the material employed was artificial culture, cardiac vegetations from natural cases in the pig, or blood from smaller animals artificially infected, and whether the material was injected under the skin or mixed with food. These results are not such as one would expect to get in dealing with the germs of a virulent contagious or infectious malady, but they are in conformity with clinical experience of the disease in this country.

It might be said that such completely negative results indicate that the disease is never transmitted by the methods employed in the experiments; but such a conclusion is inadmissible. It is impossible to doubt that the disease is caused by the bacillus first identified by Loeffler and Schütz, and it is in the highest degree probable that infection takes place by way of ingestion when the disease is naturally contracted; but, either owing to slight virulence on the part of the germ, or a natural insusceptibility on the part of pigs in this country, the disease does not assume epizootic proportions, and rarely affects more than one or two animals, even when a considerable number have been associated with those attacked and presumably exposed to the same risk of infection.

MEDICAL OFFICERS AS VETERINARY INSPECTORS.

An interesting report on the influence of milk in spreading zymotic disease has recently been contributed by Mr Ernest Hart to the columns of the *British Medical Journal*. The communication does not owe its interest to any fresh information which it imparts regarding the transmission of disease through the agency of milk, for as a matter of fact it adds nothing to our knowledge of that subject. Its chief interest lies in the very peculiar views which are here put forward with the authority that attaches to anything written on matters of public health by the editor of the *British Medical Journal*. The author of the report appears to think that a veterinary surgeon is entirely out of place when he inspects dairy cows as a precaution against the transmission of disease from which they are suffering to human beings consuming their milk. He is unable to conceive any more ridiculous enactment than that which ordains that a medical officer must be accompanied by a veterinary surgeon when he desires to inspect cows suspected of furnishing milk infective to human beings. He contends that "every medical officer of health should be armed with full power of entry on dairy premises, and of inspection of cattle where milk is regarded by him as causing disease in his district;" and he is disgusted because "we are content to go on disregarding the warnings of our State health experts, and allow veterinary authorities to pass as having a clean bill of health cows which are suffering under a malady which has been proved over and over again to be disseminating disease and death to human beings."

A layman reading these quotations would naturally feel much alarmed, and he would probably ask, what is this deadly malady spread to human beings by cows which "veterinary authorities pass as having a clean bill of health?" It need hardly be said that Mr Ernest Hart is here referring to cow-scarlatina, and that he is very angry because that disease has not yet found a place in veterinary nosology. If he really believes that the cow is a natural subject of scarlatina, and that milk furnished by cows so affected is a frequent cause of scarlatina in human beings, we cannot blame him for being angry with the veterinary profession, but he ought not to have allowed his indignation to lead him into such an untenable position as the one he appears to hold. His argument might be stated thus. Cows suffer from scarlatina, and the milk of cows so diseased causes scarlatina in human beings. Veterinary surgeons are unable to diagnose this disease in the cow, while medical officers of health are able to diagnose it; hence inspection of milch cows should be assigned to medical officers of health. We venture to submit that the premises are wrong, and that the conclusion would not be justified even if they were correct.

Do cows ever suffer from scarlatina? That is a matter of evidence, and not of faith or revelation. Some veterinary surgeons, perhaps the great majority of them, consider that the evidence in the Hendon case did not prove that the cows were affected with scarlatina, or that the milk of these cows, as it left the udder, contained the germs of scarlatina. But it is hardly fair to pretend that all medical officers of health, not to say all medical practitioners, believe in the occurrence of scarlatina in the cow. Professor Crookshank is not a medical officer of health, but we believe he is engaged in teaching many who desire to become medical officers of health, and the medical world knows his views about the Hendon disease. He, and we venture to think many other eminent medical teachers in this country, and almost the entire medical profession outside of Great Britain, absolutely disbelieve in the cow theory of scarlatina outbreaks. The veterinary profession, or such members of it as do not accept that theory, may therefore console themselves with the thought that if they are sinners in regard to this matter they have sinned in very good company.

But even supposing that some veterinary surgeons have erred in this matter, it is surely going too far to suggest that their opinions regarding the state of health of cows are henceforth and for ever to be set aside in every case in which a medical officer of health believes that disease is being conveyed from the cow to human beings by means of the milk. Mr Ernest Hart thinks that "sustained and frequent inspection of cow-sheds is called for in order that they may be kept well under supervision," and that this inspection, "must be made by those whose qualifications fit them for the duty—as, for example, medical men with special training in cow conditions and maladies in relation with disease in the human subject." It is a very pretty idea, but we fear it is not workable. Who are to give these medical aspirants to veterinary offices this "special training in cow conditions," and where is the training to be carried out? But it is unnecessary to pursue such a ridiculous proposal further. The professional education which is given to veterinary students, and the experience which is gained in veterinary practice, form the indispensable training for those who desire to inspect cattle with regard to the state of their health. When medical officers of health can agree among themselves as to the "cow conditions and maladies" that justify the condemnation of the milk they will find that veterinary inspectors are quite capable of diagnosing these abnormal states, and meanwhile they may settle down to the conviction that the public do not credit them with sufficient knowledge of animal diseases to justify them in undertaking veterinary work.

In the very number of the *British Medical Journal* which contains the report from which we have quoted we find an editorial article in which the writer comments in very indignant terms on what is supposed to be an attempt on the part of sanitary inspectors to usurp the work of medical men. Speaking of the Scottish Public Health

Bill, the article says:—"Clause 45 provides for the medical officer or any other legally qualified medical practitioner certifying as to the necessity for the cleansing and disinfecting of a house or articles or their destruction. An amendment has been put down proposing to substitute the sanitary inspector for the medical practitioner. In the frame of mind which possesses this Committee, this may very well be accepted, or even another which empowers a sanitary inspector, under a warrant, to enter any premises and examine any person. This is a very strong power to give the medical officer, but the sanitary inspector!" With very little alteration the last sentence would express our views regarding the proposal to throw upon medical men work which the law at present assigns to veterinary surgeons. To make it legal for a veterinary inspector to enter any premises and examine any cow is to give him very strong power, but a medical man!

Reviews.

Lehrbuch der vergleichenden Pathologie und Therapie des Menschen und der Hausthiere. Von Prof. Dr Georg Schneidemühl, Privatdozent der Thiermedizin an der Universität Kiel. Leipzig: W. Engelmann.

THE second and third parts of this work have now been issued. As the title of the work indicates, it deals with the pathology and therapeutics of the principal diseases of man and the domesticated animals. The second part treats of poisons—mineral, vegetable, and animal; diseases caused by animal parasites; general diseases of the blood; and diseases of the skin. The third part deals with diseases of the organs of digestion, and affections of the spleen, and partly considers diseases of the organs of respiration.

It need hardly be said that the various subjects are not treated at exhaustive length, for otherwise the work would amount to a small library in itself. At the same time a reasonable space is devoted to all diseases of importance, and etiology, symptoms, morbid anatomy, and treatment are duly discussed. As a work in which the diseases of man and those of the domesticated animals are treated in juxtaposition, it cannot fail to be interesting to both medical men and veterinary surgeons. The price of each part is six marks, and the fourth and final part is to be issued during the course of the present year.

Studien über das Kehlkopfpfeifen der Pferde. Von K. Günther, Geheimer Medizinalrath, Professor und Direktor A. D. der Thierärztlichen Hochschule zu Hannover. Zweite Auflage. Verlag der Deutschen Thierärztlichen Wochenschrift, Karlsruhe.

THE second edition of Professor Günther's monograph on "Roaring" discusses, to the length of 112 pages, the causes, course, diagnosis, and treatment of this too common defect. As is well known, the author was a pioneer in the surgical treatment of roaring, and he more than hints that some of those who within recent years have written monographs on the subject, and brought forward new (?) operations, have added little that is of practical value to what he himself made public thirty years ago. He alleges that Möller's operation in Germany has not proved more valuable as a cure for roaring than it did when carried out a few years ago on British Army horses. Tracheotomy, in Günther's opinion, is still the best operation for roaring.

A Text-book of Veterinary Ophthalmology. By Geo. G. Van Mater, M.D., D.V.S., Professor of Ophthalmology in the American Veterinary College, etc. New York: William R. Jenkins, 1897.

THE author's excuse for publishing this work is that his students have often asked him to recommend something that could be studied without necessitating the perusal of many pages. We imagine that there is no teacher living who could not furnish this excuse for the crime of writing a bad book. We do not mean to say that Dr Mater's book is in every sense a bad one, but it has one quality that is certain to irritate a purchaser, viz., that it is *not* a text-book of veterinary ophthalmology. The illustrations are nearly all of the human eye, and the text conveys the impression that the author has himself had no experience in the treatment of eye affections in the horse or other domesticated animals.

CLINICAL ARTICLES.

SOME CLINICAL CANINE NOTES.

By F. HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

OTORRHŒA (*Canker of the Ear*).

IN the Free Clinique of the College otorrhœa in the dog and cat is one of the diseases most commonly met with; occasionally the treatment presents some difficulty and is very prolonged. The chief secret of success seems to be the thoroughness with which the chosen remedy is applied.

The mischief is generally caused by something which produces irritation of the ear passage; such as eczema of the skin around the base of the ear, and the consequent constant scratching on the part of the animal; accumulation of wax, skin scales, dirt, or parasites; tumours, enlarged ceruminous glands, polypi, granulation tissue or other foreign body. In the case of dogs constantly in the water the latter may be (like any other foreign body) a predisposing cause, but the common idea that the disease is largely confined to water-dogs is certainly an erroneous one.

The symptoms are those of shaking the head, the animal often holding the latter to one side, scratching the base of the ear or the skin around it, a tendency to rub the ears against things, expression of pain at intervals by yelling or howling, and a discharge of fœtid dark-coloured material more or less thick in consistency.

When the ear is manipulated a sucking noise may often be heard, and in very severe cases deafness may be present.

The treatments adopted are various, but the foundation upon which they are all based is to allay irritation in the affected parts by cleanliness and thoroughness of application. The ears should be syringed two or three times daily, either with some warm alkaline solution (as solution of potassium carbonate, sodium carbonate, or soap and water) or a solution of spirit and water (1-4 to 1-20), the base of the ear being manipulated with the fingers, then carefully rinsed with clean water, dried by the aid of antiseptic wadding, and afterwards filled with some dry antiseptic or mild astringent powder, such as boracic acid, iodoform and starch, oxide or carbonate of zinc and starch, bismuth subnit., etc., etc.

This treatment is a very useful one, and gives a great proportion of successes; in one chronic case at the clinique during the past year we tried almost every remedy we could think of over a period of nine months without more than temporary success, the owner bringing the animal every day, when required, so that the application might be made thoroughly; but after a week of treatment by syringing with spirit and water, careful drying and then filling the ears with boracic acid, a permanent cure was effected. Of other treatments which give excellent results we have the use of watery solutions of subacetate of lead (1-2 to 1-20), sulphate of copper, sulphate, chloride, or acetate of zinc, nitrate of silver, cocaine, and many antiseptics, such as creolin, carbolic acid, etc.; whilst of oily or greasy preparations we have had liniment and glycerine of carbolic or tannic acids. When the ear cavity is almost occluded by polypi, enlarged ceruminous glands, granulation tissue, or tumours, these must be removed either by the knife, wire snare, or direct application of caustic, and the wound afterwards treated by some of the above-mentioned methods.

In some cases, too, we have found benefit from the application of some sedative (such as belladonna ointment or liniment) to the base of the ear, and in others by some stimulant such as soap liniment. Of course, if eczema is present this must be treated separately.

When parasites are the cause, a solution of camphor in olive oil or lard (1-4 to 1-8) forms a most effectual remedy for narcotising and dislodging them; creolin ointment or other parasiticides are also effectual, the ear being afterwards cleared of the dead insects by cleansing with an alkaline solution.

OPERATIVE TREATMENT FOR HERNIA.

In the *Journal of Comparative Pathology* for June 1895 nine cases of hernia, and in the *Veterinarian* for March 1896 five cases, occurring in the Free Clinique during 1894 and 1895, and treated by operative measures, are recorded.

Of these, twelve were inguinal, one abdominal, and one umbilical.

Since then the following cases have occurred:—

Case 1.—24th January 1896. Toy Manchester terrier, six weeks old, with large scrotal hernia on each side. The herniated organs were easily replaced, but soon returned. As the animal was very weak the owner was advised to postpone the operation until it was at least three months old, unless absolute necessity occurred. About a fortnight later the patient returned, the owner stating that the animal was in great pain and very weak. On account of this last condition an unfavourable prognosis was given, but the operation was, by urgent request of the owner, performed. The skin was incised under cocaine and the organs easily reduced, but the patient afterwards refused all food and died the next day.

Post-mortem revealed nothing abnormal, death being due to collapse.

CASE 2.—28th February. Fox terrier bitch, four or five years old, suffering from hernia of the pregnant uterus in the left inguinal region. Upon opening the sac the hernia was found to consist of the left horn of the uterus containing two fœtuses about four or five weeks old. The horn and contents were ligatured and removed, the inguinal ring being sutured after the stump had been returned into the abdomen.

29th February. All going on well, but on 1st March the animal became dull and refused food, a quantity of blood-stained discharge escaping from the wound.

2nd March. Animal collapsed and died quietly about 4 P.M.

Post-mortem revealed the cause of death to be metro-peritonitis, the abdomen containing a quantity of fluid which could not escape, owing to the closeness of the sutures in the inguinal ring. This suture ought to have been released and the cavity washed out as soon as symptoms of dulness appeared.

CASE 3.—15th April 1896. Manchester terrier bitch, eight years, suffering from double inguinal hernia. That on the left side was about the size of a duck's egg, that on the right quite as large as a newly-born child's head. On account of the great extent of the hernia, a very guarded prognosis was given. The right sac was first dealt with; upon incision the inguinal ring was found to be enormously dilated, the herniated organs consisting of small intestines, omentum, pancreas, bladder, right horn of the uterus, cæcum, and even a part of the rectum. Considerable difficulty was at first experienced in returning and keeping in place all the organs. The uterus was removed and the remainder returned. The inguinal ring was sutured and a drainage tube fixed in position. The hernia on the left side was not touched. During the next twenty-four hours the bitch fed well and seemed bright, but about thirty-six hours after the operation she became dull, refused food, and died.

Post-mortem revealed peritonitis to be the cause of death, the intestines being united together, and there being a large blood-clot in the abdomen. Upon noticing that she was dull a stitch had been removed from the inguinal ring, but apparently without the desired effect.

CASE 4.—16th March 1896. Irish terrier bitch, seven years, good condition, suffering from double inguinal hernia. On the left side the sac was about as large as half a cocoanut, and on the right side as big as half a walnut. It had been noticed by the owner for about twelve months. She had had a litter of puppies by a retriever about twelve months ago. When opened the left sac contained small intestines, the left horn of the uterus, and a cystic ovary about as large as a walnut. The intestine was returned, the left horn being ligatured near the body of the uterus, and excised together with the enlarged ovary. The internal ring was then sutured with one stitch, and the external ring with two stitches, the skin being sutured in the usual way. The hernia on the right side was reduced without much difficulty, and the ring sutured. After-treatment consisted in treating the wounds antiseptically, and the patient made an uninterrupted recovery.

CASE 5.—20th July 1896.—Fox terrier bitch, three and a half years, suffering from a hernia on the left side about as large as half an orange. The owner stated that she had never had pups, and that he had noticed the hernia about a year or so; it had got larger during the past three or four months, and occasionally she had been in pain. Upon opening the sac the herniated organs proved to be the uterus and omentum; the latter was almost entirely removed, and the former returned into the abdomen. A compress of antiseptic wadding was applied close to the ring in order to keep the uterus up.

21st July. A hard body which appeared to consist of some herniated organ could still be felt, and the animal was therefore again placed on the operating table and anæsthetised. This hard body proved to be the uterus; silk ligatures were passed round the body and each horn as high as possible, the whole being excised; on the right side the ovary was not secured, but on the left side it was removed. The inguinal ring was again sutured, space being left for drainage. An uninterrupted recovery took place, the animal being sent home on the 27th. I saw the case again on the 31st and on the 18th of August, when there was only a small scar left. On the 19th of October she was brought back suffering from constipation; there was no return of the hernia. She was distinctly in œstrum, and the owner said that he had noticed it for several days.

CASE 6.—27th October 1896. Persian cat, ten weeks old, male, suffering from an unsightly umbilical hernia about as large as a walnut. The defect was congenital. Reduction was easy but not permanent. The organ herniated proved to be omentum; a portion was excised, the remainder being returned into the abdomen, and the umbilical ring sutured. The animal was castrated at the same time. The wound healed well, but on the 30th of November there was still a small hernia about the size of a hazel nut, so that the operation was repeated. As on the previous occasion, the hernia consisted of omentum; a portion was again removed and the ring sutured. After this operation a perfect recovery ensued.

CASE 7.—24th February 1897. Dachshund bitch, about two years, suffering from double inguinal hernia. The animal had been noticed to suffer pain occasionally. The organ herniated proved to be uterus, the right and left horns being through their respective inguinal rings. An incision was made on the left side only, the right hernia being reduced by aid of the finger introduced into the abdominal cavity. Unfortunately the animal died four days later of acute pneumonia accompanying distemper, showing decided signs of illness on the day after the operation. I afterwards found out that her appetite had been irregular, and that she had had a slight nasal discharge for several days previous to the time of operating.

CASE 8.—1st March 1897. Fox terrier, one year old, suffering from umbilical hernia about as large as half a walnut. It proved to consist of omentum, and was excised, the umbilical ring being sutured. The patient made an uninterrupted recovery.

CASES 9 and 10.—13th April 1897. St. Bernard pups, three months old, from the same litter, in which five out of six were similarly affected. In each case the hernia was about as large as an orange. The herniated organ was omentum, and was in each case firmly adherent to the umbilical ring; it was dissected off and returned, the umbilical ring being sutured. In each case the stitches gave way and the hernia returned again as large as before; the owner would not trouble to have one of them operated upon again, but the second one was operated upon by Mr Woollatt of Class D, and made a good recovery.

CASE 11.—12th June 1897. Schipperke bitch, eight years old, suffering severely from asthma; the hernia, which was a double inguinal one, was about as large as a large walnut on each side, and had been noticed about three weeks. Reduction in each case was effected without incising the hernial sac, the skin alone being cut, and the rings sutured in the usual way.

13th and 14th June. Going on well, slight sanguineous discharge. This case has since made a good recovery, the wounds being almost healed (23rd June).

CASE 12.—10th June 1897. Bull bitch, eight years old, suffering from an inguinal hernia on the left side about the size of a goose egg. This had been noticed for about a month, and appeared to be getting larger. The animal was chloroformed and the skin incised; an attempt was made to return the contents without opening the hernial sac. but without complete success, and eventually an incision was made into it. The herniated organ proved to be uterus; this was returned and the inguinal ring sutured. The skin was afterwards sewn up, and drainage provided for. The case progressed favourably and the bitch made an uninterrupted recovery, being sent home at the end of a week with only a small wound to be attended to.

In each of the above cases the method of operation was to fix the animal on its back on an operating table, apply some local or general anæsthetic, cut down upon the herniated sac or its contents, return the organs into the abdomen after removing any found necessary, and suture the inguinal ring and skin.

After-treatment consisted either in applying iodoform and collodion (1 in 10) or dry antiseptic wadding and bandage, keeping the parts clean and the patient at rest under as good hygienic arrangements as possible. Attention must always be paid to the sutures in the ring, as they must be removed upon the slightest sign of peritonitis in order to allow pus to escape. Summing up the twenty-six cases, and taking into consideration the condition of the patients that died, I still think that the results are very favourable, and that this operation of cutting down upon the hernial sac, returning the organs, and applying sutures, offers the best prospects to the surgeon.

LAPAROTOMY.

Case A.—*Stricture of Colon.* 23rd Nov. 1896. Fox terrier dog, two years old, suffering from extreme emaciation and weakness. Suspecting the cause to be abdominal tumours, the patient was anæsthetised and laparotomy performed. The cause was found to be stricture of the colon, just where it joins the rectum. The strictured portion was quite 2 inches long, and its walls were enormously thickened; the colon itself was dilated to three or four times its normal size. The diseased portion was removed, and the cut edges of the gut were sutured together. The animal died within twenty-four hours after the operation; the extreme debility and the extent of the lesion made the case quite a hopeless one.

Case B.—*Lympho-Sarcomata.* 16th Nov. 1896. Spaniel, eighteen months old, very weak and emaciated. Suspecting abdominal tumours to be the cause, laparotomy was performed; the operation revealed a large number of tumours on the intestine, varying in size from a hazel nut to an orange. As it was impossible to remove them the anæsthetic was forced and the animal destroyed. Prof. M'Fadyean examined the tumours, and declared them to be lympho-sarcomata.

Case C.—27th Nov. 1896. Was similar to Case B but occurred in a cat; the animal was very much exhausted and very weak when brought. Laparotomy revealed enlargement of the intestinal lymphatic glands.

Case D.—12th March 1896. Pomeranian bitch, four or five years old, suffering from constipation and vomiting of fœtid material, very weak and ill, had been so for about a week. Suspecting an intussusception, or at all events some foreign body blocking up the intestinal canal, laparotomy was performed under chloroform; no foreign body or intussusception could be discovered, nor indeed anything to account for the symptoms set up. The wound was therefore closed and bandaged, half a grain of morphia being injected subcutaneously on two separate occasions. The next day the animal seemed very much weaker and would not eat, dying quietly from syncope during the night. *Post-mortem* examination revealed nothing abnormal.

It is impossible, of course, to form any conclusions from the above cases, as the weak state of the patient or the great extent of the lesion made a successful result almost impossible; but that the abdomen of the cat or dog is a very good field for surgery is well shown by the success that follows ovarian and uterine operations.

EXCISION OF THE RECTUM AFTER PROTRUSION.

In cases of prolapse of the rectum excision is adopted as a last resource, if all attempts to retain it in position prove to be futile.

The operation is performed under some anæsthetic, local or general, and consists in passing a probe (a thermometer case answers very well) up the centre of the bowel, then suturing several times through the bowel on to this close to the anus, in order to prevent it from disappearing completely into the abdomen when the everted portion is cut off, so as to keep the two cut surfaces in apposition. After this the everted portion is cut completely through, one part being removed, and the other part allowed to slowly find its way into the abdominal cavity.

CASE 1.—19th Feb. 1896. Cat, female. Rectum had been out nine days. Excised under ether. The animal died two days later, but a *post-mortem* was not obtainable.

CASE 2.—1st March 1896. Cat, female, about two years. Rectum had been out three-and-a-half days. Excised under cocaine. This case made a good recovery.

CASE 3.—18th Feb. 1897. Cat. Rectum had been everted nearly three weeks. Excised under cocaine. This patient went on well for four days afterwards, keeping bright, and promising to make a good recovery, but at the end of that time she died suddenly. The owner suspected poison, but unfortunately no *post-mortem* was made.

CASE 4 was a very interesting one. The first operation occurred 25th February 1897. The patient was a bull terrier about eighteen months old; the rectum had been everted for two days, and had been replaced and kept in position by sutures, which eventually gave way. Excised under chloroform, about 2 inches being removed. Opium was administered internally, and the animal kept as quiet as possible.

9th March. About 1 inch which still projected was excised under cocaine, and on 23rd March a still further portion of about half-an-inch under a mixture of cocaine and eucaine.

About ten minutes after the dog strained and put out quite 5 inches of intestine through the anal opening; this was returned and the anus sutured across. Gave 30 grains of extract of cannabis indica in pill.

24th March. Gave 10 grains of extr. cannabis; animal straining occasionally, but not sufficient to expel the gut. After this matters progressed favourably, and the dog made a good recovery.

ORCHITIS.

Orchitis in the dog is not very commonly met with. The patient in this case, a mastiff six or seven years old, was suffering from unilateral orchitis, and was brought to the college on 7th January. No history of injury was obtainable; the owner stating that he had noticed the swelling for about three weeks. The testicle now was fully six or eight times its normal size, cool and firm to the touch, and not giving the animal any apparent pain. Castration was performed, and with the exception of some little trouble owing to an abscess in the groin, the dog made a good recovery.

OVARIOTOMY.

Ovariotomy, or "spaying," is an operation which deserves a great deal more attention from veterinary surgeons than it usually receives. It is one which in the bitch and cat can usually command a fair fee, and as a surgical operation it is one of the neatest, giving the operator every facility for displaying the value of antiseptic and abdominal surgery. The patient should be placed under an anæsthetic and fixed in the dorsal position with legs well extended; hair should be removed by shaving around and over the seat of operation, and the parts rendered thoroughly aseptic. An incision is then made either in the flank, a little to the side of the mammæ, or else in the median line (the latter is the one I prefer usually), through the skin and abdominal muscles, bleeding being arrested by artery forceps or tampons of dry antiseptic wadding; a director inserted enables one to operate with less risk of injuring the abdominal organs. The ncision should be about half-an-inch to an inch long, and commence directly over the pelvic border. At this stage a blunt probe or flexible catheter should be inserted into the vagina towards the womb, the fore-finger or middle-finger of the right hand being passed into the abdomen. The left hand, or an assistant, carefully moves the probe about, so that the right hand has no difficulty in feeling it inside the body, and thus distinguishing uterus from intestine; the finger is then lightly hooked round it and some part of the uterus drawn towards the opening. Having recognised the uterus, carefully manipulate each horn so as to expose the ovary, and remove the latter either by mere incision after ligature or by scraping. If a ligature be used, aseptic catgut is certainly to be preferred to silk. The muscular wound is then drawn together loosely with interrupted catgut suture, and the skin wound with a couple of continuous sutures, the whole being dried and painted with iodoform and collodion (1-10), space being left for drainage if thought necessary. If everything has been aseptic the wound will heal in three or four days, and as a rule the animal does not even lose its appetite. Aseptic wadding and bandages are sometimes necessary if the wound breaks open, and a broad leather collar is placed round the neck if the animal persists in attempting to lick it.

The following cases have been operated upon as above described:—

CASE 1.—19th February 1896. Collie bitch, eight months old. Operation performed in the flank, only ovaries removed. This animal never showed any abnormality, feeding well all the time and making a rapid recovery.

CASE 2.—29th July 1896. Pug bitch, about eighteen months old, suffering from ulcerating papilloma of the vagina. Ovariotomy was performed in the hope that it would lessen the irritation produced in the vagina at the period of œstrum. The operation was performed on 30th July, the parts being afterwards bandaged; on the 3rd August it was found necessary to release some of the stitches and give free vent to a quantity of pus, the wound in the abdomen also being reopened. After this the animal made an uninterrupted recovery, being sent home on the 6th.

CASE 3.—16th August 1896. Fox terrier bitch. Ovariotomy under chloroform. All went on well, and the animal was sent home on the 22nd.

CASE 4.—12th October 1896. Kitten, nine weeks old. Anæsthetised with A. C. E. mixture. The wound in this case was made in the median line, and had quite healed on the 15th, the animal being sent home on the 19th.

CASE 5.—16th November 1896. Bull bitch, with ulcerating papilloma of vagina. Ovariotomy was performed with the same object as in Case 2, the median line being the site chosen, and the wound healed without any bad symptom.

CASE 6.—24th November 1896. French poodle with ulcerating papilloma of vagina. Ovariotomy was performed with the same object as in Cases 2 and 5. In this case the incision was made in the flank; the wound had not been kept quite aseptic and a certain amount of pus formed, but eventually this all became discharged and the animal made a good recovery. On the second day it was necessary to wash out the abdomen with creolin and water and insert a drainage tube; this was removed four days later and the wound allowed to close.

CASE 7.—17th January 1897. Cat, one-and-a-half years old. The median line was chosen as the site, and the wound healed without any sign of suppuration, the animal being kept in the infirmary for seven days.

CASE 8.—25th January 1897. Irish terrier bitch, eight or nine months old. The incision was made on the median line, and the wound healed rapidly, the patient being sent away ten days later.

CASES 9 and 10.—May 1897. Cat, two years old; fox terrier bitch, three or four years old; the latter was in œstrum at the time. Both were done on the median line, and in each case the wound healed without formation of pus, the animals being sent away in a week.

CASE 11.—May 1897. Cat, two years old. The incision was made in the flank, but owing to some neglect of antiseptic precautions a lot of pus formed, and the abdomen had to be re-opened and drained. Eventually, however, the animal made a good recovery.

CASE 12.—21st June. Kitten, ten weeks old. The incision was made on the median line, the ovaries only being taken away. The animal kept bright throughout, and made a rapid recovery.

CASE 13.—For this I am indebted to Mr Walder of Class D, who operated during the Whitsuntide vacation. The patient was a terrier, aged two years, in good condition. The operation was performed on the median line, and the wound had healed in about ten days.

CASE 14.—15th June. Kitten, four months old. The median line was chosen as the seat of operation, ether being used as the anæsthetic. The wound was dressed with iodoform and collodion (1–10) and healed in three days, the appetite remaining good throughout.

CASE 15.—16th June. Kitten, between two and three months old. The particulars of the operation were identical with those of Case 14, and the animal made a good recovery.

HYSTERECTOMY.

In the *Journal of Comparative Pathology* for June 1896, four cases of this operation are recorded. The method of procedure is much the same as with ovariotomy, except that a part of the uterus and both horns are removed instead of merely the ovaries.

CASE 1.—On the 8th of June I was intending to perform ovariotomy upon a Persian cat, ten months old, when I found upon incising the abdomen that she was pregnant; one horn contained two fœtuses, and the other contained one, about three or four weeks old. The method of procedure adopted was to place a catgut ligature around the body of the uterus and also one round each of the horns above the ovary, and excise the intervening portion.

The external wound was treated antiseptically, and, although the animal was somewhat dull and off her feed, she eventually made a good recovery.

CASE 2.—For this I am indebted to Mr Walder, of Class D, who performed the operation during the Whitsuntide vacation. The patient was a spaniel, aged three years, in œstrum. The seat of operation was the median line, the ovaries and horns being ligatured with aseptic catgut and removed; the horns were taken off just below their junction with the body. The wound suppurated, but eventually the animal made a good recovery.

CASE 3.—15th June. Cat, aged about two years. The whole of the uterus from above the ovaries to the commencement of the body was removed, the contents consisting of three fœtuses between seven and eight weeks old. Aseptic catgut ligatures were placed above each ovary and around the body of the uterus before incision. The external wound, made in the median line, was sutured and dressed with iodoform and collodion, and had quite healed in three days, the appetite remaining good throughout.

JAUNDICE IN THE DOG, AND OTHER CASES.

By P. S. HOWARD, M.R.C.V.S., Royal Veterinary College, London.

CASE I. *Jaundice and Intussusception in the Dog.*—An aged fox-terrier dog was brought to the Clinique suffering from jaundice, the visible mucous membranes being very yellow in appearance.

In the treatment doses of calomel (gr. ij.) were administered once a day in the form of a pill; a light nutritious diet was also prescribed. At the expiration of a week the patient seemed to be recovering, the yellowness of the membranes being less marked, and the appetite showing signs of improvement.

The same treatment was continued during the second week, at the end of which a relapse occurred and the animal died.

On making a *post-mortem* examination the liver was found to be congested, the gall bladder contained a small quantity of bile, and the common duct was not markedly distended. In the first portion of the small intestine, about 8 inches from the pylorus, there was an intussusception 3½ inches long; from its appearance the lesion must have been recent, and therefore was probably not the cause of the jaundice.

CASE II. *Rupture of Liver in the Dog.*—The body of an average sized bull-terrier dog, about three years old, was brought to the Clinique for *post-mortem* examination.

The history was that the dog had suddenly died without any symptoms having been noticed.

It was observed that the visible mucous membranes were abnormally pallid in appearance. When the abdomen was incised there was an escape of clotted blood, and on further examination the abdominal cavity was found to be filled with the same material.

A rupture with lacerated edges was present at the lower border of the liver in close proximity to the portal fissure; a clot of blood occupied the space between the ruptured surfaces.

The liver was in a state of advanced fatty infiltration, and this probably predisposed to the rupture; at the same time no direct evidence of a blow or other exciting cause was obtainable.

CASE III. *Polypus in Eustachian Tube of the Cat.*—A cat about eleven years old was brought to be destroyed.

The owner stated that the cat had been sneezing constantly for nine months and was gradually becoming worse.

The head was sawn in halves after death, and a growth about the size of a small pea was discovered projecting into the pharynx from the left Eustachian tube.

On slitting up the tube the pedicle was found to be attached to it about half an inch from the pharyngeal opening.

It may be mentioned that the ear of the same side was badly affected with otorrhœa, but on following up the Eustachian tube to the middle ear no connection between the two conditions could be discovered.

CASE IV. *Sarcoptic Mange in the Fowl.*—A fowl was brought with a scaly affection of the legs, and also to a slight extent of the head.

The lesions consisted of crusts from half an inch to two-thirds of an inch in thickness; the exterior of these was dry and grey in colour, while their interior was yellow and moist. On closer inspection their surface showed a number of very minute granules about the size of a pin's point.

When the scales were stripped off the leg a bleeding surface was exposed. The scales covered the foot and the whole of that portion of the leg which was devoid of feathers.

On examining the scales microscopically numerous parasites were found; these were identified as belonging to the genus sarcoptes. The females are oval in shape, and their legs are so short as to appear like small dots around the margin of the parasite. The two posterior pairs of legs do not project from beneath the parasite at all. The males are about quarter the size of the females, with much longer legs in proportion to their size.

Their movements are nothing like so rapid as in many other varieties of the sarcoptes; they resemble the other varieties of this genus also in that the females burrow, while the males are found exclusively on the surface of the crusts.

The treatment of this disease must be both preventive and curative; in the former it is necessary to isolate all the affected fowls, and in the latter to deal with the lesions, this being quite successful if actively carried out. The scales should be removed by either peeling them off, or better still by soaking them in warm water and soap or some alkaline solution, such as carbonate of soda, after which one of the ordinary dressings for mange should be applied.

Since the above case came into the Clinique, I have seen several additional ones, although they have not been so severe in nature. The disease seems to be quite familiar to fowl-keepers, although, at the same time, its true nature is not generally recognised by them.

PERONEAL TENECTOMY FOR THE RELIEF OF STRINGHALT.

By William Robb, F.R.C.V.S., Glasgow.

Although unable to show an extensive list of successful cases, I am induced by the favourable results in the two cases here noted, to ask others to give this operation a trial before condemning it. The operation is of the simplest character, consisting in the removal of a portion of the peroneus tendon below the hock. In stringhalt the balance of power would seem to be with the flexors. The peroneus is a flexor of the hock, and while the loss of it may balance the power of flexors and extensors, yet it is not sufficient to interfere with the animal's action. By this I mean that if one interfered with the flexor metatarsi as I shall describe, I am afraid the cure would be worse than the disease, leaving the balance of power with the extensors.

Case I. The first animal I operated on was a van gelding, which on becoming unfit for that purpose had been used as a tracer. Owing to the further advancement of this affection he became totally unable to trace, and I asked to be allowed to operate. Having cast him, I clipped and washed the skin over the tendon, and then cut right down on the top of it. The tendon was then cut through close to the end of the metatarsus, disected out of its bed of tissue, and severed from its union to the extensor pedis. The other leg was operated on in the same way. The wounds were stitched up and dressed and the animal put into slings. He was very stiff for a few days, but this soon disappeared, and on the wounds healing he went to work. It is about eighteen months since this was done, and he kept fit for work until a few days ago, when he received a kick on the hock and had to be destroyed. In one leg not the slightest sign of stringhalt could be detected, and only occasionally in the other. Now this was no slight case of stringhalt, but an animal who flexed his hocks completely at every step.

Case II. The other case while flexing his hocks abducted his legs slightly at the same time. He became quite unfit for light lorry and van work, and on another animal being got to take his place I operated as described above. The result has been very gratifying; only at times is there any suspicion of his old complaint. His condition may be judged from the fact that immediately after his wounds had healed he had to go back to his former work, owing to the new purchase taking pneumonia. He now trots freely, a gait that was for some time impossible, owing to the exaggerated action of his hind limbs.

Report.

CLINICAL HISTORY OF CASES OF SWINE ERYSIPELAS[1] (ENDOCARDITIS).

CASE A.—In April 1896 a brood sow in young was bought by A., and a fortnight later she farrowed seven.

Nine weeks later the bonhams were sold as follows:—

One to B.; two to C.; two to D.; and two to someone unknown.

The brood sow was subsequently sold fat. A. had no swine then remaining.

On or about the 20th August 1896 B.'s pig (a store sow) appeared to be suffering from a cold; and on the 27th August 1896 it developed violent symptoms of sickness, and was reported on that date as affected with swine fever. The symptoms were described to the police as "refusing food." B. had two other swine (a sow and a hog) on his premises at that time, bought four months before. The departmental veterinary inspector visited on the 29th August 1896; described the symptoms presented by the reported sow as follows:—

"Very much discoloured, the whole back and sides being of a livid blue colour. When made to move, the animal emitted great cries of pain. Temperature $104\frac{4}{5}^\circ$ F."

This sow was slaughtered, and viscera sent to department for *post-mortem* examination. The heart showed vegetations on the mitral valve, and on being sent to Professor M'Fadyean he reported having found numerous swine-erysipelas bacilli therein.

Veterinary inspector revisited on the 9th September 1896, but could not detect any appearances of ill-health among B.'s remaining two swine.

On the 17th September 1896 visited the premises of C. and D. with the following result: C. had sold the two swine referred to "fat" three weeks previously. The two bought by D. were still on his premises, and were found to all appearances healthy and thriving well.

On the same date, 17th September 1896, the premises of B. were again visited. The hog was found in good condition, and apparently in good health, temperature $102\frac{3}{5}^\circ$ F., but the sow was not putting on condition, temperature $103\frac{1}{5}^\circ$ F. It was also observed that "the tail was hanging down limp like the lash of a whip."

This last-mentioned animal was slaughtered on the 23rd September 1896, and the viscera forwarded for examination. Heart and viscera were examined and found free from disease.

CASE B.—About the middle of February 1896 a sow, about eight weeks in young, was purchased at a fair, and brought on to premises on which there were already four store swine. (These latter were sold fat about May.) On or about 12th April 1896 the sow gave birth to seven young; four died shortly after birth—some at least having been killed by the sow. The three remaining (stores) on being weaned were housed in a sty and not allowed out afterwards.

On the 26th August 1896, midday, the owner, noticed that the stores had not eaten their usual quantity of food. One of them was breathing heavily (abdominal), and had a slight redness over back of neck and back of ears. This animal was found dead in the afternoon.

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[1] From the Second Report of the Departmental Committee appointed by the Board of Agriculture to inquire into the Etiology, Pathology, and Morbid Anatomy of the Diseases classed as Swine Fever, 1897. Eyre & Spottiswoode, London.

On the 30th August 1896, the remaining two were noticed ill in a similar way, and were reported on same date. One was slaughtered on the 31st August 1896, the other on the 11th September 1896. Result, "Not swine fever," but as the hearts showed vegetation on the mitral valves, they were sent to Professor M'Fadyean, who found swine-erysipelas bacilli in both cases.

The symptoms presented by the above three stores were similar, and as follows:—

Off feeding; disinclined to move about; redness of skin.

The animal referred to above as having been slaughtered on the 11th September 1896 was at the time of the inspector's visit (same date) considered by the owner to be getting better, as it had returned to its feeding. The inspector found it not inclined to move much; breathing hurried and laboured; a diffused redness of the skin about back of neck and ears; bowel excretion normal.

The sow gave birth to another litter (10) about the 10th September 1895. Both sow and young were inspected on 3rd October 1896 and appeared healthy. No other swine on premises.

Case C.—A sow and a hog were bought by A., from some person unknown, at a fair in the month of May 1896. A. had also bought a sow a month earlier from B.

The first-mentioned sow became sick on the 4th September 1896 and died on the 7th. While sick the following symptoms were noticed:—

"Complete loss of appetite, great difficulty in breathing, moaning, and livid discoloration along abdomen, around neck, and along ears."

The viscera were sent to Dublin for *post-mortem* examination. Result, "Not swine fever."

The heart showed vegetations on the mitral valve, and was sent to Professor M'Fadyean, who found swine-erysipelas bacilli.

Departmental inspector visited A.'s premises on the 8th October 1896, and found the hog and the remaining sow apparently in health.

Also visited B.'s premises, and found three pigs of the same brood as A.'s other sow; also their dam and three bonhams. All these appeared to be in health.

Case D.—The owner in this case purchased two swine (a hog and a sow) in April 1896. No other swine on premises then, or since.

These two swine remained in apparent good health up to about the 29th September 1896, when one of them (the hog) became ill, and was reported to the police on the 3rd October 1896 as supposed to be suffering from swine fever.

Symptoms were:—Loss of appetite, ears purple coloured, purple patches on body, coughing, breathing rapidly, straggling gait, and weakness of the hind extremity.

The pig was slaughtered on the 7th October 1896, and viscera sent up for *post-mortem* examination. Result, "Not swine fever."

The heart showed vegetations on the mitral valve, and, on being sent to Professor M'Fadyean, was found to contain swine-erysipelas bacilli.

The remaining sow had not been ill at any time while on the premises, and was sold at a fair on the 1st October 1896 (apparently for slaughter).

Case E.—A pig jobber purchased three swine at a fair on the 3rd July 1896; seller unknown. These animals were sold on the same day—

One to A.; one to B.; one to some person unknown.

The pig referred to as sold to A. appeared healthy for about a week, when it got drowsy and lost its appetite for a few days. It then got better again and ate fairly well, but did not seem to thrive. The owner noticed it again ill on or about the 4th September 1896, with its sides panting and its ears becoming bluish-red in colour; it lay still in the sty. Next day he found it dead, with further discoloration around the neck.

The viscera were forwarded to Dublin for *post-mortem* examination. Result, "Not swine-fever." The heart showed vegetations on the mitral valve, and was sent to Professor M'Fadyean, who found therein swine-erysipelas bacilli.

The pig referred to as sold to B. showed no signs of any ailment. The owner fattened it, and sold it to a dealer.

There are no swine now remaining on A.'s premises.

CASE F.—The owner of the pig connected with this case bought the animal (a hog) at a fair in the month of June 1896, from someone unknown to him.

The hog showed signs of sickness about a fortnight previous to the report being made (the 1st September 1896), and exhibited the following symptoms:—

The appetite became impaired, and finally was lost. There was a slight discharge from the eyes, and the breathing was short and hurried. The ears became pendulous, and the animal was observed to cough frequently.

Our inspector was assured that there was no discoloration apparent until after death, which occurred on the 5th September 1896.

The viscera were found free from swine fever, but the heart showed vegetations on the mitral valve, and on being forwarded to Professor M'Fadyean, he found swine-erysipelas bacilli therein.

This was the only pig the owner possessed, but there were two other swine in adjoining styes which frequently came in contact with it. One was bought in a fair in March 1896, and the other in July 1896, both from persons unknown.

These two swine were inspected on the 7th October 1896, and they were apparently in health, one of them being nearly fat.

CASE G.—Six swine were sold on or about the 1st April 1896.

Four were bought by A.; two were bought by B.

A. fattened and sold these four in a fair about the 7th July 1896. He informed our inspector that they were never at any time ill, and always took their food.

B. stated that her purchases were good feeders but bad thrivers, and in consequence she sold them on the 1st August 1896 to C. They also thrived very badly with C., and finally one was found dead on the 9th September 1896.

Our inspector could get no history as to any illness prior to its death, the attendant stating that they were never sick.

The heart of this pig showed vegetations on the mitral valve, and was sent to Professor M'Fadyean. He found swine-erysipelas bacilli.

One hog remained on C.'s premises. It was inspected on the 12th September and 5th October 1896, and found to all appearances healthy.

CASE J.—The pig (a sow) connected with this case was bought about June 1895 with a companion sucker. The companion was kept until fat and then sold some six months later.

The sow had nine bonhams on the 13th June 1896. Seven of these were sold in August; purchaser not traced; the other two (hogs) are now on owner's premises, and when inspected by a departmental inspector on the 2nd October 1896 seemed healthy.

About the 5th September 1896, the sow was noticed ailing, and she died on the 8th of same month. The symptoms observed were:—

"Disinclination to move, refused to take food, constipation, skin became red at back of ears."

Result of *post-mortem* examination, "Not swine fever."

The heart showed vegetations on the mitral valve, and was sent to Professor M'Fadyean, who found swine-erysipelas bacilli.

CASE L.—Two swine (a hog and a sow) were purchased about 10th April 1896 by A. from B., who live in adjoining houses.

Early in August 1896, A. observed the two swine "refuse to feed for a few days."

The sow continued slightly cramped in the hind limbs, and became sick again about 1st September 1896.

On 8th September 1896, A. reported this animal to the police as suspected of being affected with swine fever, and on 11th September 1896 the departmental veterinary inspector visited the premises and had her slaughtered.

The symptoms he observed were:—Difficult breathing, fits of coughing, as if trying to expel some foreign body from trachea and larynx; salivation and champing of jaws; weakness of limbs, particularly the hind ones; and, when not walking, resting on the quarters like a dog; pallor of skin; and excessive thirst. Temperature 103⅘° F.

The viscera were sent up to Dublin for examination, and were found free from swine fever, but the heart showed vegetations on the mitral valve, and on being forwarded to Professor M'Fadyean he reported having found the bacilli of swine erysipelas.

The veterinary inspector revisited on 9th October 1896, reported the remaining pig (the hog) as having become affected in the legs, and walking with difficulty, sitting on quarters like a dog, but otherwise apparently healthy.

This animal was slaughtered on 20th October 1896, but the viscera (including the heart) were healthy.

The swine belonging to B. were inspected on 9th October 1896, and they were reported healthy.

CASE M.—Three swine were bought in a fair on the 19th July 1896, from a pig dealer, who had himself bought them the day before from a farmer at another fair.

The three swine were of the same litter, and were bought as follows:—

Two (a hog and a sow) by A.; one (a hog) by B.

A. and B. kept their swine in adjoining yards; the former had only the two mentioned, and the latter has two others (sows).

Four days after their purchase A.'s (two) swine went off their feed, appeared dull, and a diffuse rash came out all over the body, but most marked on the abdomen, perineum, thighs, and roots of ears. This rash disappeared in two days after the attack, and the pigs began to feed again, but became lame or crippled and moved with difficulty.

Nothing more was noticed (with the exception of the lameness, which continued) until the 3rd September 1896. On that date the sow, on being let out for a run, fell down, frothed at the mouth, breathed heavily, but after a time got better. It refused food, however, was inclined to lie, and when the owner tried to raise it on its feet it uttered painful grunts.

On the 9th September 1896 it was found dead in the sty.

Viscera were "Not swine fever" on *post-mortem* examination, but heart showed vegetations on the mitral valve, and on being forwarded was found by Professor M'Fadyean to contain the bacilli of swine erysipelas.

The departmental veterinary inspector at the time of his visit, 9th October

1896, found A.'s remaining pig lame in both hind legs, and swollen from the hocks downwards, but otherwise it appeared in good health. Age about eight months, weight 10 stone.

The hog belonging to B. (of the same litter as A.'s) was also lame in both hind legs, and much smaller than the other two swine on B.'s premises, though of the same age, and getting similar feeding.

The inspector was instructed to have these two slaughtered, and the viscera forwarded for *post-mortem* examination.

This was done (20th October 1896). The *post-mortem* examination showed that both sets of viscera were healthy.

CASE Q.—Nine swine were bought by a dealer in a country fair on the 21st July 1896, from a seller unknown. Four of these were sold (22nd July 1896) to a city swine keeper, one of the four being the pig directly connected with this case.

This pig was noticed drowsy on the morning of the 21st September 1896, and refused to eat its food. Discoloration of the skin was not noticed. It died that night.

Viscera did not show swine fever.

The heart showed vegetations on the mitral valve, and on being sent to Professor M'Fadyean he found swine-erysipelas bacilli therein.

The three other swine (stores, about five months old) did not show any sickness since they were purchased.

The pig that died was apparently in health until 21st September 1896.

CASE R.—A pig dealer bought twenty-four pigs at a fair early in June 1896, and subsequently disposed of them as follows:—

Twelve to persons unknown, sold the same day in the fair; ten taken to a fair a few days later, six of these were sold to persons unknown, and four to A.; two to B. on the 7th June 1896.

It was not possible to state definitely from whom the two sold to B. were purchased at the fair, as the twenty-four were obtained from three different sellers and mixed. These two swine were the first lot kept by B., and there were not any swine kept in the immediate neighbourhood of his premises since these swine were bought.

On 11th August 1896 B. reported the two swine (one hog, one sow) to the police as affected with swine fever. The symptoms were described as "purple blotches through them, and refusing to eat." The hog died, and its viscera were forwarded to the department for *post-mortem* examination. Result, "Not swine-fever." The inspector to the Local Authority reported the other ill. He described the symptoms as "off feed, poor, blotches over body, temperature 105° F."

The departmental inspector visited on 21st August 1896, and inspected the remaining sow, which he considered healthy.

The animal improved considerably up to 20th September 1896, when it again showed symptoms of disease, but in a less aggravated form than before.

On the 22nd September 1896 the owner reported the sow again; same symptoms as given above. The local authority inspector visited and slaughtered it. He described its symptoms as "animal very dejected; accelerated breathing; large purple patches over body, particularly at the back of the ears and shoulder." He also observed desquamation of the cuticle and loss of hair on the places where the purple patches had been. The viscera were sent to the department. Result of *post-mortem* examination "Not swine-fever."

The heart showed vegetations on the mitral valve, and was forwarded o Professor M'Fadyean, who found swine-erysipelas bacilli therein.

A departmental inspector visited A.'s premises on 26th September 1896, inspected the four swine, which he declared healthy. From inquiries they would appear to have been healthy since purchase.

CASE S.—The pig in this case was bred upon the owner's premises and was nine or ten months old. It was reported to the police on 22nd September 1896 as suspected of being affected with swine fever. The first symptoms of sickness were observed the preceding evening, 21st September 1896. They were as follows:—

"High temperature, viz., 104·5° F., cramped legs, red skin in spots, almost unable to walk, loss of appetite, extreme prostration, loss of flesh, isolation on part of the animal, skin very hot on abdomen, vesication."

Another was reported as similarly affected.

This animal was slaughtered by inspector to Local Authority on 23rd September 1896, and viscera sent to department for *post-mortem* examination. Result, "Not swine-fever." The heart showed vegetations on the mitral valve, and was sent to Professor M'Fadyean, who found swine-erysipelas bacilli.

A departmental veterinary inspector visited the premises on 29th September 1896, and found thereon:

One brood sow and six bonhans. Seven stores. Five fat pigs.

The five last mentioned were of the same litter as the pig slaughtered on 23rd September 1896, and the other reported ill was also one of this lot. The inspector reported that this was showing decided symptoms of sickness, as follows:

"When made to move it emitted cries of pain, there was considerable stiffness in the legs, particularly the hind legs. The head was allowed to hang down, the ears being pendulous. Appetite very irregular, animal losing condition."

This pig was slaughtered on 8th October 1896, and on the viscera (with heart) being examined they were found healthy.

The seven stores above referred to were brought on to the premises about two months before the inspector's visit (on 29th September 1896). They were apparently in health, and he was informed that none of them had shown any sign of illness since their purchase.

The sow had been sent some distance for service about five months previously. Four swine had been sold fat about three months before.

No other movements of swine for a very considerable period.

CASE T.—The pig in this case (a hog) was bred by A., and sold at a fair to B. in May 1896. The following were also sold by A. the same day:—

One to C.; one to D.

The breeder referred to (A.) stated that this hog had not shown any signs of illness up to date of its sale. After coming into B.'s possession, it kept in apparent good health up to the 24th July 1896, when it became ill, and was reported to the police on the 27th July 1896. The symptoms then were:—

Dulness, not feeding, thirst, continually lying on its litter in the sty, and showing disinclination to move."

It recovered in four or five days. Was inspected by an departmental veterinary inspector on 30th July 1896, who found it "free from swine fever."

About the 11th September 1896 it became ill again, but was not reported to the police until the 22nd September 1896. It was slaughtered on the 24th September 1896, and viscera sent up for *post-mortem* examination. Result, "Not swine-fever;" but there were vegetations on the mitral valve of the heart, and on that organ being forwarded to Professor M'Fadyean, he found swine-erysipelas bacilli.

The symptoms observed during the animal's second illness, were:—

"Loss of appetite, cough, rapid and difficult breathing, ears purple-coloured, diffuse purple patches on the body, principally along the abdomen, on the shoulders and neck, weakness of the hind extremity, with straggling gait."

B. had had no other swine than this hog on his premises for more than two years.

The two swine sold to C. and D. were inspected on the 10th October 1896, and found healthy. A.'s premises were visited on same date, and the swine thereon were also found in health. A. informed our inspector that he never had any sickness among his pigs.

Case U.—The pig (a hog) connected with this case (together with a sow) was purchased at a market on the 6th January 1896. At this time the owner did not keep any other swine, and none were brought on to his premises since.

The hog was noticed to be ill on the night of the 19th September 1896.

Symptoms presented were:—

"Animal was not feeding; then it turned red at back of neck, and was disinclined to move, and weak behind."

It died on the morning of the 22nd September 1896, and viscera were forwarded to Dublin for *post-mortem* examination.

Result, "Not swine-fever."

The heart showed vegetations on the mitral valve, and was sent on to Professor M'Fadyean, who found the bacilli of swine erysipelas.

The remaining sow was inspected on the 6th October 1896 by a departmental inspector, who reported that the animal seemed healthy.

Case V.—Six swine were sold at a fair on the 19th August 1896, as follows:—

Four to a person living in the neighbourhood of the fair, who, however, could not be traced.

One to A.; one to B. These were about four months old when purchased.

The pig (a hog) purchased by A. did not show any signs of illness until the 18th September 1896. It was then taken suddenly ill, in fact it was let out of the sty in the morning, and galloped down the field, but on returning it fell down before regaining the sty. From that time until the date of its slaughter, 23rd September 1896, it would not feed properly, but was kept alive on a little gruel. Slight discoloration at roots of ears and quickened respiration were also noticed by owner, but there was no rash seen since the pig was purchased.

The hog was slaughtered by veterinary inspector to Local Authority on the 23rd September 1896, and viscera forwarded for *post-mortem* examination. Result, "Not swine-fever." The heart showed vegetations on the mitral valve, and was sent on to Professor M'Fadyean, who found swine-erysipelas bacilli.

The local inspector described the symptoms as follows:—

"Staggering gait, slight discoloration at roots of ears, which may have been due to lifting pig by those organs; back arched; breathing hurried; pendulous abdomen; knuckling over on hind fetlocks; animal weak and depressed, but no evidence of pain."

B.'s pig (a sow) was visited by departmental veterinary inspector on 7th October 1896, and found in health. He was informed that she had not been unwell since time of purchase.

Case W.—Two swine (a hog and sow) were purchased from a dealer, who had bought them from some one unknown. Seven weeks later, the 27th September 1896, the owner reported the sow to the police as affected with swine fever. These two were the only swine kept.

Three or four days before report the pig was noticed off her feed and lazy; these symptoms gradually became more marked, until she refused to eat and would only take liquids. She lay persistently, but could walk when roused. No knuckling over on fetlocks, but evidently great weakness. Constipation marked, but relieved after a time by physic. Urine darker and denser than normal. The ears and hind quarters became of a scarlet and purple hue.

The sow was slaughtered on 28th September 1896, and viscera sent to department for *post-mortem* examination. Result, "Not swine fever."

The heart showed vegetations on the mitral valve, and on being sent on to Professor M'Fadyean was found to contain the bacilli of swine erysipelas.

The hog showed no symptoms of sickness, and was apparently healthy and in fair condition at the time of the departmental inspector's visit, the 13th October 1896

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THE EXAMINATION OF COW'S MILK FOR THE DETECTION OF PATHOGENIC PROPERTIES.

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(*Continued from p.* 156)

8. HAVING given a general account of the procedure I adopted for the examination of a first series of thirty-six samples of mixed milk, and also explained the method followed in recording some of the results obtained, I may now, without entering into similar details again, give the results of other series of examinations conducted in exactly the same way.

Nine mixed milks received from Liverpool,[1] completing the first set of forty-five specimens, gave the following results.

Two samples produced well-marked tuberculosis, and in one case also severe septic lesions. Four (including the last) produced severe septic infection, more specially when injected into the peritoneum. Two of these specimens produced acute purulent peritonitis, fatal in twenty-four hours; and one produced extensive necrosis and suppuration when injected subcutaneously. Two samples produced slight inflammatory lesions, indicating irritating properties. Only one was entirely free of any noxious properties.

As in the previous cases of rapidly fatal septic infection, it was possible to isolate from the peritoneal exudation and from the blood of the heart a bacillus to which further allusion will be made later on, and in one case the streptococcus pyogenes was also found in the blood. In addition to these two organisms the local lesions several times contained the staphylococcus pyogenes aureus.

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[1] These nine specimens are not included in the list of specimens given on the second page of this paper. If these nine specimens and a new series of thirty-two samples referred to later on be added to the first list, it will be found that the total number of samples is 111 instead of 70.

9. To sum up the points brought out by the examination of the series of forty-five mixed milks, the following statements may be made.

(1.) They were all more or less contaminated with extraneous matter, animal and vegetable, evidently derived from the cow, the stable, milk pails, cans, etc.

(2.) Cells from the udder were present in all cases, but sometimes they were very few in number, at other times they were excessively abundant. The milks which proved by inoculation to be tuberculous contained in all cases a large number of cells, but other milks containing many cells produced no tuberculosis, and sometimes even not more inflammation than milks containing very few cells.[1]

(3.) Twenty-nine out of the forty-five specimens contained a large number of bacteria of various kinds.[2]

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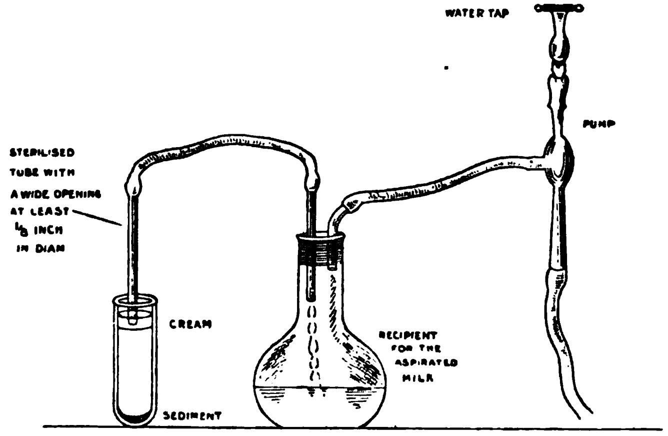
[1] The cells present were chiefly large and small epithelial cells, vacuolated or not; debris of these cells were very frequent, and often resembled leucocytes. Leucocytes were present in a little more than half the cases, but were very seldom numerous. A few large epidermal scales were seen in about half the cases.

[2] As the organisms present were not isolated and cultivated it is difficult to speak positively regarding their identity, but I made careful microscopical examination in all cases, and the following types of microbes were found. (1) Staphylococci almost always. (2) Streptococci in less than half the cases. (3) Diplococci in about half the cases. (4) Tetrades or sarcinæ in about one-fifth of the cases. (5) A bacillus having all the morphological features of the bacillus lacticus was invariably present and sometimes excessively abundant; in five or six cases, however, it was very scanty. (6) Another short bacillus resembling the bacillus coli was present in many cases. (7) Long slender bacilli were not very frequent. (8) Thick long bacilli resembling the bacillus subtilis were found in nearly half the cases, but were seldom numerous. (9) Yeasts and Oidia were also found in a number of cases, but they were seldom abundant.

I have in the first part of this paper omitted accidentally to mention the method which I used in preparing specimens of milk for microscopical examination.

After the milk has been centrifugalised, the sediment is separated by removal of the supernatant fluid; this is done by aspirating the fluid by means of an air pump so as not to disturb the sediment. This may seem an unnecessary complication, but anybody who has tried to obtain a sediment free from cream after milk has been centrifugalised must have found that it is not at all easy to aspirate or syphon off the layer of cream, which is very dense and resistant. Having exposed the sediment, a sample is taken with the sterilised platinum loop and spread over a cover-glass. The film is allowed to dry, and immediately after fixed in the flame of a Bunsen burner in the usual way. The cover-glass is then placed in a small covered capsule into which is poured about ¼ to ½ ounce of absolute ether and absolute alcohol (equal parts), in this it is left for from two hours (at least) to twenty-four hours, after which the covered capsule is placed in a shallow dish full of very hot water, so as to bring the ether to the boiling point or near it. After one-quarter to half-an-hour the cover-glasses are taken out, well washed with absolute


WATER TAP
PUMP
STERILISED TUBE WITH A WIDE OPENING AT LEAST ⅛ INCH IN DIAM
CREAM
SEDIMENT
RECIPIENT FOR THE ASPIRATED MILK

(4.) The reaction, notwithstanding the large number of bacteria present in the majority of specimens, was amphoterous in thirty-two cases; in six cases it was acid, and in seven it was alkaline. (As six out of these seven specimens were sent to the laboratory together, it is probable that the alkalinity was due to some of the alkali used in cleaning the bottles not having been entirely removed by subsequent washing. I may say, however, that all the specimens received in the laboratory had evidently been collected with great care and uniformity of method, and that the inspector whose duty it was to collect the samples had taken all the trouble necessary to obtain clean bottles.)

(5.) Notwithstanding the large number of bacteria often present in the milk, the sediment of seventeen samples could be injected into the subcutaneous tissue and the peritoneal cavity of guinea-pigs without producing any lesions beyond a slight temporary irritation not interfering with the general health of the animal; this was ascertained by careful daily observations and weighing in a number of cases.

In seventeen other cases marked inflammatory lesions were found at the seat of inoculation or in the lymphatic ganglia connected with the inflamed part, and in many instances the general health of the animal suffered, as indicated by a rapid loss of weight. This illness, however, was temporary, and the animals allowed to live long enough recovered. In a few cases, however, the lymphatic ganglia connected with the seat of inoculation remained large. These enlarged glands differed from tuberculous glands in that they contained no tubercle bacilli, were fleshy, had a brownish red colour, and showed no necrotic areas, clearly visible. A short bacillus could be demonstrated in several of these enlarged glands.

Eight of the milks produced intense septic lesions in the shape of acute fibrino-purulent peritonitis or extensive subcutaneous suppurations and necrosis.

In six of these cases the blood of the heart was examined bacteriologically, and a bacillus closely resembling the bacillus coli communis was found invariably present; in one of these cases the streptococcus pyogenes was also isolated. The same bacillus, associated or not with the streptococcus pyogenes and the staphylococcus pyogenes aureus, was also isolated from some of the local lesions. The bacillus, after being cultivated in a state of purity for several generations on agar, produced when injected into guinea-pigs a rapidly fatal septicæmia associated with peritonitis, just as when the milk had been injected.

alcohol, and stained in various ways. Tubercle bacilli can be beautifully stained by the Ziehl-Neelsen method, all the fat having been dissolved and washed out. The methylene blue brings out the cells and bacteria other than the tubercle bacillus. If such a film be stained directly with carbol fuchsin, the coagulated proteids occupying the space between the now dissolved fat globules, stain deeply, so that a permanent preparation showing the original size of the fat globules can be obtained. Such preparations are not suitable for the study of cells or micro-organisms. If, however, before staining with carbol fuchsin the specimen is left for a short time in 10 per cent. sulphuric acid, the proteid layer does not stain any more, and the cells and micro-organisms stain readily. Films of the sediment, cream, and milk half-way between the layer of cream and the sediment were repeatedly examined to ascertain whether the solid impurities were completely forced into the sediment. It was found that the sediment contained the bulk of these extraneous products, but many bacilli, cells, and sometimes even foreign bodies, such as hairs, were carried up by the cream. In two tuberculous cases tubercle bacilli though most abundant in the sediment were also easily demonstrated in the cream; the intermediate layer of separated milk was always freest from all impurities. Bacteria were naturally much more numerous in specimens which had taken a long time to reach the laboratory and in those that had been exposed to a high temperature than in the others. Specimens which had been kept for two or three days in winter often contained less bacteria than specimens examined in spring or summer.

The bacillus was present both in the local lesions and in the blood. I examined the blood of the heart of four guinea-pigs which had not shown sign of disease after being inoculated with milk, and also the blood of a guinea-pig that had become tuberculous, and in none of these cases could I obtain any organism, the tubes inoculated with large quantities of blood remaining absolutely sterile.

Finally, three of the samples examined produced tuberculosis on the animals inoculated.

(6.) None of the milks which produced tuberculosis contained enough bacilli to make their detection possible by microscopical examination of a reasonable duration. The bacilli could not be detected by the examination of two drops of sediment prepared in such a way that these organisms should have been easily recognised. The inoculation test proved, however, that in two of these specimens the bacilli were numerous enough in the sediment of 1½ ounce of milk to produce well-marked tuberculosis in forty-two days and eighty-six days respectively. In the third case the bacilli must have been very few, for only one of the two animals inoculated contracted the disease, and the lesions remained almost enterely localised in the lymphatic ganglia immediately connected with the part inoculated; the bacilli were very few in these lesions.

General Results of the Examination of Mixed Milks.—To conclude this part of the inquiry, the results may be tabulated as follows:[1]—

Properties of forty-five samples of mixed milks kept for twenty-four to fifty-six hours (one seventy-eight hours) after milking.

1. Samples producing no noxious effect or only trivial transient lesions	17
2. Samples producing local irritation, but no general infection	17
3. Samples producing intense local irritation, and general septic infection	8
4. Samples producing tuberculosis	3
Total	45

REPORT ON SPECIMENS OF UNMIXED MILKS COLLECTED IN MANCHESTER DIRECTLY FROM THE UDDER IN STERILISED VESSELS.

THE nature of the first Manchester investigation[2] differs considerably from that of the Liverpool one.

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[1] In another series of thirty-two mixed milks examined for the health office in Manchester, I have obtained similar results, but as they have not been officially reported upon I will use them only with reference to the part of this communication which has no bearings on the official investigation.

[2] This part of the enquiry is based on the examination of the milk of thirty-one cows, most of which were tuberculous. With two exceptions all these cows were kept in Manchester or its suburbs. Twenty-four specimens were sent to me by Dr Niven in order that I should examine them for tuberculosis. Ten other specimens (from seven different cows) were collected specially for me. In three cases the milk of the same cow was examined both before and after the injection of tuberculin. The lungs and other organs of several of the tuberculous cows, the milk of which had been obtained previously, were examined in the laboratory after the animal had been slaughtered and the diagnosis made by the veterinary surgeons confirmed. The udders of seven tuberculous cows were also examined in the laboratory; two of these from which infective milk had been obtained were proved to be tuberculous both by microscopical examination and by inoculation experiments, five were either healthy or showed non-tuberculous lesions, and three of them on inoculation produced no effect.

The object which Dr Niven had in submitting specimens to me for bacteriological investigation was to ascertain to what extent the milk of cows found by the veterinary inspector in Manchester to be affected with advanced tuberculosis, was capable of producing tuberculosis.

Taking advantage of the material sent to me I determined to extend the investigation. Dr Niven who has, for a considerable time, paid much attention to the influence which milk has on the production of disease, gave me every facility to obtain the information I required.

Mr King, the Chief Veterinary Inspector, showed himself most willing to help me, and I have to thank him and his assistants (more especially Mr Holburn), for the trouble they have taken in this matter.

1. To collect the facts I wanted I had a form prepared in which all particulars regarding specimens sent to me were entered at the time of collecting the samples. The facsimile of this form will show the nature of the information which I thought desirable to obtain, and will indicate at the same time how the facts alluded to further on were obtained.

This label is to be fixed to each Sample of Milk sent to the Bacteriological Laboratory for Analysis, and all the information obtainable entered under the following heads.

Name of Farm Lab. M. No.
Situation
Description of Cow
No. of Cows kept in same Byre
Byre:—Clean, Not Clean; Ventilated: Well, Badly; Lighted: Well, Badly.
Hands of milker:—Clean, Not Clean. Teats:—Clean, Not Clean.
Does the Cow show any evidence of Disease? Where?
Has the Cow been submitted to tuberculin test? Reaction.
Nature of food General: Yes or No.
Quantity of Milk yielded Local: Yes or No.
Date of Milking hour A.—P.M. Signature of
Bottling Sample *id* A.—P.M. Inspector
Bottle sterilised or not

N.B.—Bottle, which should hold 8 ozs., can be sterilised by boiling half-hour in water after cleaning. Corks should be also sterilised.

2. The accompanying table will give the main results of the examination of twenty-seven samples of milk obtained from twenty-four cows. (The milk of seven other cows was also examined, but as the examination was not carried out in the same way, the results would not have been strictly comparable, and for this reason they have been excluded from the list, but the results obtained with them were quite in accordance with those I make use of here.)

From this table it appears that the milk of six out of the twenty-four cows was capable of producing tuberculosis. Seven specimens produced marked local irritation, in one case very severe but no general infection, no specimen produced rapidly fatal infection, eleven milks produced no marked irritation.

3. It is important to remember, in interpreting these resu (1) all these cows were, with the exception of one, suspected c culosis, (2) most of them were in an advanced state of tuberculo (3) only seven of them had apparently healthy udders, and remainder ten had certainly diseased udders. It was inte that such a number of diseased cows had been selected, and not be correct to assume that the results obtained repre average state of things; this being well understood the resu now be analysed.

4. The first question which suggests itself is, *What relation between the state of the udder and the pathogenic propertie milk?*

The results of inoculations will I think show that, ge speaking, the milk from diseased udders was the only one of producing tuberculosis, but that with regard to common lesions the difference between the milk from diseased and udders was not very striking; this will be shown by the fo tables:

Pathogenic Properties of Milk from Diseased and Healthy Ud

Milk from Udders.		*Producing Tuberculosis.*				*Producing marked Irritation.*				*Producing marked Irri*		
		Actual No.		*Percentages.*		*Actual No.*		*Percentages.*		*Actual No.*		*Perce*
A. Certainly diseased	10	5	6	50·0	31·5	3	5	30·0	25·30	2	8	20·0
B. Probably diseased	9	1		11·1		2		22·2		6		66·6
C. Healthy	5	0		0	0	2		40·0	40·0	3		60·0
Total .	24	6				7				11		

Pathogenic Properties of Non-tuberculous Milk from Diseased and Healthy Udders.

Milk from Udders.			*Producing marked Irritation.*				*Producing no mar Irritation.*			
			Actual No.		*Percentages.*		*Actual No.*		*Percent*	
A. Certainly diseased	5	13	3	5	60·0	38·5	2	8	46·0	6
B. Probably diseased	8		2		25·0		6		75·0	
C. Healthy . .	5		2		40·0	40·0	3		60·0	6
Total .	18		7				11			

S (WITH
INOCULATION.
ved during Life
found P.-M.
ecovery
arrhœa—
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tion—recovery
mmation—
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artial recovery
ion well-mark
tion—recovery
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berculosis
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n
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c glands
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P.M.
n—recover
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S (WITH THE EXCEPTION OF ONE).

INOCULATION.		
ved during Life found P.-M.	*Tubercle Bacilli in Lesions.*	REMARKS.
–	—	Milk coagulated; experiment failed
ecovery	0	Milk coagulated
	0	
arrhœa—	0	Milk coagulated
ecovery	0	Milk coagulated
	—	id
tion—recovery	0	Slight local induration at seat of inoculation
	—	
–	—	
ammation—	—	Notwithstanding the great length of time this milk had been kept it was quite sweet
ammation—	—	Milk taken just before tuberculin test
	—	id 14 hours after injection of tuberculin
artial recovery	—	Before injection of tuberculin
——	—	14 hours after tuberculin
ion well-marked	—	Before injection of tuberculin
tion—recovery	—	14 hours after tuberculin
on	—	
berculosis	Many	External evidences of tuberculosis were clear 9 days after inoculation of animals
	Abundant	
on	—	
on	0	
culosis	Abundant	The calf of this cow was killed on the seventh day, and was found to have tuberculous lesions of the mesenteric glands, liver, and lungs. All these lesions were examined in the laboratory. The cow when slaughtered was found in a state of advanced tuberculosis, thoracic and abdominal, and one of the uterine cornua was distinctly tuberculous
of peritoneum c glands	Not found	
culosis	Moderate	Milk taken during the tuberculin reaction
	id	
lammation and rgement	0	Milk taken during the tuberculin reaction
ndular enlarge-	0	Milk taken during the tuberculin reaction
	—	
ppuration and rgement	0	
eginning and	Few	Milk collected in the cistern, opened aseptically, only 2 ccs. of milk obtained
erculosis	Many	id
on	0	
P.M.	—	
	—	
n—recovery	—	
	—	
	--	

on. These samples have been collected in thirteen different byres.
, whilst the cows were under the influence of tuberculin.

In comparing the results of inoculation of milk showing distinct evidence of disease of the udder by the presence of a large number of cells and more especially pus corpuscles, with the effects of inoculation of milk showing no such evidence of disease, similarly ambiguous results are obtained with regard to non-tuberculous milks.

Pathogenic Properties of Milk, Rich and Poor in Cells from the Udder.

	Producing Tuberculosis.		*Producing marked Irritation.*		*Producing no marked Irritation.*	
	Actual No.	*Per cent.*	*Actual No.*	*Per cent.*	*Actual No.*	*Per cent.*
Cells abundant or very abundant	6	46·2	3	23·1	4	30·8
Cells scanty or in moderate numbers	0	0	2	25·0	6	75·0

This table would seem to show that about half of the milks containing such a large number of cells that the existence of mastitis could hardly be doubted were tuberculous, but of the remaining half the majority had no noxious properties whatever.

Of the milks which did not exhibit any trace of disease of the udder none were found tuberculous, and one-quarter were capable of producing irritation. If we exclude tuberculosis the influence of disease of the udder on the production of irritative lesions will be more exactly ascertained.

Pathogenic Properties of Non-tuberculous Milk.

	Producing marked Irritation.		*Producing no marked Irritation.*	
	Actual No.	*Per cent.*	*Actual No.*	*Per cent.*
Cells abundant . .	3	42·9	4	56·2
Cells scanty or moderate	2	25·0	6	75·0

Disease of the udder seems therefore to account for the noxious properties of certain samples of milks, yet when other possible factors are considered, it will be evident that other causes are at work, and to determine exactly the possible share taken by disease of the udder (apart from tuberculosis) as a source of human disease, it will be necessary to compare only specimens which have been examined immediately after their being drawn from the udder.

5. Further doubts are thrown upon the primary importance of non-tuberculous disease of the udder by a comparison of the effects of mixed and unmixed milks. If these results be tabulated we get the following figures.

Mixed Milks from the Town Market, and Unmixed Milks from Diseased Cows Compared.

Milk Producing.	*Mixed Milks.*		*Unmixed Milks.*	
	Actual No.	*Percentage.*	*Actual No.*	*Percentage.*
No marked irritation . . .	17	37·74	11	45·76
Marked local irritation, but no general infection	17	37·74	7	29·12
Intense local irritation and general infection	8	17·76	0	0
Tuberculosis	3	6·66	6	24·96
Total .	45	99·90	24	99·84

The most striking outcome of this comparison is that even *the milk of markedly diseased cows is much less often capable of producing irritation than the ordinary mixed milk supplied for consumption*, and whilst 17 per cent. of the latter are capable of producing severe infection, rapidly fatal, none of the milks obtained direct from the udder, in sterilised vessels, produced any affection of comparable intensity.

Even with regard to the minor forms of irritation, such as local abscesses, inflammatory enlargement of lymphatic ganglia, mixed milk proved inferior in quality to unmixed milk from diseased cows.

The same difference is also shown by the comparatively small number of mixed milks which were free from all noxious properties, for whilst only 37·74 per cent. of the mixed milks proved perfectly innocuous, 45·76 (*i.e.* 8 per cent. more) of the unmixed milks showed the same quality. If it be remembered that twenty-three out of the twenty-four of the latter had been taken from tuberculous cows, and that more than one-half of these cows had diseased udders, it will be evident that to get a proper idea of the relative noxiousness of mixed and unmixed milks on the basis of the results quoted above, the tuberculous milks should be excluded.

If this be done there remain forty-two specimens of non-tuberculous mixed milks, and eighteen of non-tuberculous unmixed milks. The pathogenic properties of which were as follows.

	Not Irritating.	*Irritating locally.*	*Highly Virulent.*
	Per cent.	*Per cent.*	*Per cent.*
Mixed milks . . .	40·8	40·8	19·2
Unmixed milks . .	60·5	38·5	0

The obvious conclusion from this is that, *apart from tuberculous mastitis, disease of the udder cannot be the chief source of the pathogenic properties of bad milk.*

6. On the other hand, it is equally evident that something must occur

to the *milk after it has left the udder* to account for the marked difference in the properties of mixed and unmixed milks. Comparing the fate of samples which had been collected straight from the udder and of those which had been collected at railway stations, or in town dairies, it is at once evident that, after leaving the udder, the former had come in contact with nothing but sterilised bottles; in addition to this, it was ascertained in each case that the teats of the cow and the hands of the milker were clean.

In the case of the mixed milks nothing is known of the chances of contamination due to dirty udders, hands, pails, cans, etc. From the cans they were taken with due precaution to avoid any further contamination, but nothing more could be done. These specimens of mixed milks were, when collected, exactly in the state in which they would have reached households, but they had not suffered from any of the additional contaminations which might have occurred at the hands of the retailer, or in the households of the consumers.

Many of the samples were kept in the sterilised bottles for many hours; but this occurred equally in the case of the mixed and of the unmixed milks, so that any differences which were detected in the laboratory were due less to the changes taking place after the taking of the samples than to what was already present in the milk when the samples were taken.

7. It is well known that milk obtained at the farm, in the usual way, is always more or less contaminated with microbes, and that these multiply very rapidly at the ordinary temperature all the year round, much more rapidly, of course, during warm than during cold weather.

We have numerous observations recorded giving the number of microbes which may be found in ordinary dairy milk collected in the usual way.

Gösta Grotenfelt[1] gives several figures which will prove interesting in connection with the points.

Samples taken in a pasture on a fresh somewhat damp summer morning:—

No. of Bacteria in 1 *cc.*

Immediately after Milking.	*Half-hour after.*	*Two Hours after.*
10	88	1530

Samples taken in a farm at the end of the winter and under favourable conditions:—

No. of Bacteria in 1 *cc.*

Immediately after Milking.	*Half-hour after.*	*Two Hours after.*
106	980	3655

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[1] The Principles of Dairy Practice from a Bacteriological Point of View. Translation by F. W. Woll, 1894. New York, p. 83 and following.

Samples taken in byres where conditions were less favourable:—

No. of Bacteria in 1 *cc. Half-hour after Milking.*
4,100
2,450
1,890
14,670
830
3,030
5,430
21,700
1,030

Other observers have given much higher figures. Cnopf and Escherich in Munich report having found in a sample of milk recently drawn under precautions said to be of great cleanliness 60,000 to 100,000 bacteria per cc.; but I doubt whether much importance can be attatched to these numbers, because undoubtedly some unsuspected source of bacterial contamination must have existed in that case.

De Freudenreich has given very interesting results showing the rapid multiplication of bacteria in milk at various temperatures. The following table gives the results of examination made at Rütti, and is quoted by Grotenfelt.

Increase in Bacterial Contents in Milk.

	When kept at 15° *C.*	*When kept at* 25° *C.*	*When kept at* 35° *C.*
On arrival at lab.	9·300 per cc.	9·300 per cc.	9·300 per cc.
3 hours later .	1·06 times more	2·0 times more	4·0 times more
6 hours later .	2·5 times more	18·5 times more	1·290 times more
9 hours later .	5·0 times more	107·5 times more	3·794 times more
24 hours later .	163·0 times more	62,097·0 times more	5·376 times more

To have some terms of comparison as to the original bacterial contents of milk obtained in Manchester under the condition of my experiments, I had milk collected from two cows, one kept in a town byre and the other in a suburban farm. The milk was drawn from the udder directly into sterilised beakers and brought at once to the laboratory; both specimens were collected on the 12th of December 1896. The cowsheds, cows, and milkers were clean, and the cows healthy.

	Time after Milking.	*Temp. of Milk.*	*Sp. Gr.*	*Reaction.*	*Microscopical Examinations.**		*No. of Bacteria in 1 cc. Plate Culture.*
					Cells.	*Microbes.*	
Town byre milk	20 mins.	30° C.	1025	Amphot.	Very few	Very few*	2·929
Suburban byre	7 hours	17° C.	1026	Amphot.	id	Few*	90·216

* These terms correspond to the terms used in the tables to indicate the number of microbes recognised by microscopical examination.

When milk is collected in filthy, dark, and altogether insanitary byres the number of bacteria becomes excessive; in one case Grotenfelt found in 1 cc. of milk, three-quarters of an hour after milking, 67,000 bacteria.

I might quote many other figures, some of which are still more remarkable, but I have thought it better to mention only those which, being in accordance with my own observations, seem *to me* the most reliable.

I might mention in connection with this that by keeping milk at a temperature of about 4° C. I have been able to satisfy myself of the fact that instead of an increase there is, generally, a decrease in the number of bacteria, which gets more marked as the milk is kept longer.

From all this it seems most probable that (1) dirty byres, cow-keepers, cows, milk pails and cans; (2) keeping the milk in hot places and during hot weather; (3) keeping the milk for a long time before use, are the chief causes of the noxious properties too frequently acquired by that fluid.

8. I have already explained that in the case of the mixed milks it is impossible to obtain accurate data regarding the first group of causes.

With regard to twenty of the cases of unmixed milks which I examined, I was able to obtain from the veterinary inspectors accurate data as to the state of the byres and cows from which the milk had been drawn. Some of the byres were badly lighted and ventilated, but *in every case* the byre, hands of the milker, and teats, were clean, so that I could find nothing in that direction to account for the marked differences observed in the results of the inoculations.

That all the milks get infected with germs at the time of milking is certain, but no special source of gross contamination could be detected in any of the cases under examination, therefore there remained only the two factors, temperature and time, which could be investigated with any chance of obtaining a clue to the source of the irritating properties of some samples.

A few noxious germs introduced at the time of milking would, possibly, not be numerous enough to produce any pathogenic effect at first, but if allowed to multiply they might become capable of doing much harm.

This line of investigation was also indicated by the marked differences between specimens collected in summer and in winter.

In the following tables I have arranged all the specimens examined, and which had not proved tuberculous, in three groups; the innocuous, the irritating, and the virulent, in the same way as I have done in the previous tables. In each case I give also the length of time the specimens have been kept from the time of milking to that of examination, and the external temperature observed in Manchester during that time.

The recorded temperature which has seemed to me of most importance for this purpose was that observed in the shade, for such temperature would affect equally both the mixed and unmixed specimens. It is probable that many of the mixed specimens had been exposed to higher temperatures during their transit by rail, for, whether conveyed in covered vans or open trucks, they would suffer from the effects of sunshine; I found it, however, impossible to take an accurate account of this difference.

The mean between the maximum and minimum temperature in the shade has been taken for each day. When the specimen had been kept only ten hours the temperature of the day on which the examination was made is given. When kept twenty-four hours, the temperature of the previous day. When forty-eight hours, the average temperature of the two previous days. When seventy-two hours, that of the three previous days.

In every case the recorded temperature has been taken from the very complete and useful table published by Dr Niven in the "Weekly Return of the Medical Officer of Health" Manchester.

Unmixed Milks which did not produce Tuberculosis.

	No. of Reference.	*Date when Examined.*	*How long Kept.*	*Temp. in the Shade.*	*Reaction of Milk.*
			Hours.	*Average.*	
I. Milks causing no lesion, or only trivial irritation, at the seat of inoculation or near it, in one or both of the animals inoculated.	335	7 X 96	24	44·2	Amphot.
	338	12 X 96	72	46·5	„
	339A1	12 X 96	39	43·8	„
	339A2	12 X 96	25	38·0	„
	340A2	12 X 96	25	38·0	„
	341A2	12 X 96	25	38·0	„
	351	4 XI 96	24	39·3	„
	404	9 II 97	10	44·0	Slightly alkaline
	407	13 II 97	under 30	39·8	Alkaline
	408	13 II 97	30	39·8	„
	424	24 III 97	24	50·7	Slightly alkaline
	425	„	under 24	50·7	„
	426	„	„ 24	50·7	„
	427	„	„ 24	50·7	„
Averages		October November February March	28	43·8	Amphot.
II. Milks producing marked local irritation with no general infection.	326	13 VII 96	66	64·4	Strongly acid
	327	„	66	64·4	„
	357	11 IX 96	72	62·5	„
	340A1	12 X 96	39	43·8	Slightly acid
	341A1	„	39	43·8	Amphot.
	409	24 II 97	24	49·9	„
In one of the two animals death on the sixth day, but only local suppuration found *P.-M.*	423	23 III 97	90	50·0	Alkaline
Averages		July September October February March	56	47·0	Acid

Note 1.—The milk of three cows—339, 340, and 341—having been examined twice at various intervals after milking, the results of the two examinations have been entered separately. It will be noticed that in two of these cases—340 and 341—the samples kept thirty-nine hours proved more noxious than those kept twenty-five hours.

Note 2.—The milk in case 423 came from a very diseased udder, as proved by the examination of the veterinary surgeon and the examination of the sample of milk by myself; this sample was crowded with cells, and its specific gravity was only 1019.

Mixed Milks which did not produce Tuberculosis.

	No. of Reference.	*Date when Examined.*	*How long Kept.*	*Temp. in the Shade.*	*Reaction of Milk.*
			Hours.	*Average.*	
I. Milks causing no lesion, or only trivial inflammation at or near the seat of inoculation.	360	23 XII 96	52	35·5	Amphoterous
	361	„	78	35·3	„
	362	„	53	35·5	„
	374	31 XII 96	29	43·1	(?) „
	375	„	29	43·1	„
	378	16 I 97	42	35·9	„
	380	„	40	35·9	„
	381	„	42	35·9	„
	382	„	50	35·9	Slightly acid
	385	23 I 97	48	34·4	Slightly alkaline
	386	„	50	34·4	„
	388	„	50	34·4	„
	389	„	40	34·4	„
	390	„	50	34·4	„
	395	28 I 97	42	31·9	„
	398	„	40	31·9	Amphot.
	449	4 V 97	45	47·4	„
Averages		December January	46	36·4	Amphot.
II. Milks producing marked local irritation, but no general infection.	359	23 XII 96	54	35·5	Amphot.
	363	„	53	35·5	„
	365	„	27	35·5	„
	366	„	27 No certain data.	35·5	„
	368	„	27	35·5	„
	371	31 XII 96	28	43·1	„
	373	„	30	43·1	„
	376	„	30	43·1	„
	377	16 I 97	41	35·9	„
	379	„	40	35·9	„
	387	23 I 97	51	34·4	Slightly alkaline
	394	30 I 97	49	33·2	Amphot.
	396	„	49	33·2	Acid
	397	„	42	33·2	Amphot.
	401	„	28	33·2	„
	446	5 V 97	56	46·7	Acid
	447	„	45	46·5	Amphot.
Averages		December January	42	37·6	Amphot.
III. Milk producing intense local inflammation and general infection rapidly fatal.	364	23 XII 96	53	35·5	Amphot.
	367	„	27? *Not certain.*	35·5	„
	400	2 II 97	27	36·7	„
	402	„	29	36·7	„
	445	5 V 97	56	46·7	„
	448	„	45	46·5	„
	481	18 V 97	32	56·5	„
	483	„	32	56·5	Acid
Averages		February May	39	43·8	Amphot.

Second Series of Mixed Milks examined in Manchester (this Series has not been included in any of the previous Tables).

	No. of Reference.	*Date when Examined.*	*How long Kept.*	*Temp. in the Shade.*	*Reaction of Milk.*
			Hours.	*Average.*	
I and II. Cases producing no general infection rapidly fatal.	470	2 VI 97	31	58·5	?
	471	"	31	58·5	?
	472	"	31	58·5	?
	473	"	31	58·5	?
	495	7 VII 97	9.30	53·7	Slightly alkaline
	497	14 VII 97	8	64·3	Alkaline
	498	"	8	64·3	Amphot.
	500	"	8	64·3	Slightly acid
	507	28 VII 97	8.20	59·7	Amphot.
	508	"	8.35	59·7	"
	509	"	9.30	59·7	"
	510	"	9.45	59·7	"
	515	4 VIII 97	8.50	75·3	"
	516	"	9.20	75·3	"
	520	11VIII97	8.40	60·7	"
	521	"	8.20	60·7	"
	522	"	9	60·7	"
	523	"	9.20	60·7	"
	525	18VIII97	9.35	60·3	"
	526	"	9.55	60·3	"
	527	"	10	60·3	"
	528	"	10.15	60·3	"
Averages			13	62·0	Amphot.
III. Cases producing intense local lesions and general infection.	441	23 IV 97	over 36	55·3	Slightly alkaline
	502	21 VII 97	17	61·8	Slightly acid
	503	"	17	61·8	Slightly alkaline
	504	"	17	61·8	Slightly acid
	505	"	17	61·8	Acid
	517	4 VIII 97	8	75·3	Amphot.
	518	"	8	75·3	"
Averages			17	64·7	Acid or alkaline

[TABLES—*continued.*

These tables may be summarised as follows:—

I. *Mixed Milks coming from a distance of generally over* 40 *miles, and generally kept for from twenty-four to sixty hours, and even more in a few cases.* (*Tuberculous samples excluded.*)

Mean Temperature in the Shade (Manchester) during Time the Specimens were kept.	*Specimens producing no Noxious Effects.*	*Noxious Specimens.*	*Totals.*	*Percentage of Good Specimens.*
30° to 35	7	5	12	58·0
35 to 40	7	11	18	38·5
40 to 45	2	3	5	40·0
45 to 50	1	4	5	20·0
50 to 55	—	—	—	—
55 to 60	0	2	2	0·0
	17	25	42	39·0

II. *Mixed Milks coming from a short distance (generally under* 20 *miles), most of them kept for less than ten hours (with the exception of five out of the seven bad specimens, and four out of the twenty-two good specimens).* (*Tuberculous samples excluded.*)

Mean Temperature in the Shade (Manchester) during Time the Specimens were kept.	*Specimens producing no Noxious Effects.*	*Noxious Specimens.*	*Totals.*	*Percentage of Good Specimens.*
50° to 55	1	0	1	100·0
55 to 60	8	1	9	88·8
60 to 65	11	4	15	73·2
65 to 70	—	—	—	—
70 to 75	2	2	4	50·0
	22	7	29	75·68

III. *Unmixed Milks kept for various lengths of time, but collected from the Udder in Sterilised vessels.* (*Tuberculous samples excluded.*)

Mean Temperatare in the Shade (Manchester) during Time the Specimens were kept.	*Specimens producing no Noxious Effects.*	*Noxious Specimens.*	*Totals.*	*Percentage of Good Specimens.*
35° to 40	6	0	6	100·0
40 to 45	3	2	5	60·0
45 to 50	5	2	7	71·5
50 to 55	—	—	—	—
55 to 60	—	—	—	—
60 to 65	0	3	3	0·0
	14	7	21	67·2

The influence of time is well shown by the number of specimens remaining good even at high temperature when the milk had been kept only half-a-day.

On the other hand, the influence of temperature is still more evident, for in every category the number of good specimens is almost inversely proportional to the height of the temperature.

When the clear relation existing between time of keeping *plus* temperature and the noxious properties of a certain number of samples of milk is contrasted with the ambiguous results obtained when an attempt is made to connect these noxious properties with disease of the udder, it is difficult not to feel convinced that *infection of the milk outside the udder and the conditions under which it is kept are the most important factors causing it to acquire at times pathogenic properties.*

9. I have been able to collect some facts during the last few years in connection with outbreaks of epidemic diarrhœa due to the consumption of milk. I have found a close resemblance between the infection which I have produced in guinea-pigs by the inoculation of milk which had given rise to one of these outbreaks and that produced in guinea-pigs by the injection of several samples of mixed milks obtained on the market. This has led me to suspect that milk must be one of the most potent causes of the summer diarrhœa of children, an opinion which is also held by several well-known authorities. My belief has been strengthened by the fact that from all the cases of fatal septicæmia due to milk injections I have been able to isolate a bacillus identical with that which I have obtained twice from milk causing intense diarrhœa in children and a few adults. Other organisms have been present in a few specimens, but the only organism constantly present has been this bacillus. This microbe retains its virulent properties even after being cultivated for several generations outside the body. It resembles closely the bacillus coli communis in its pathogenic action, its mode of growth on gelatine, agar, potato, milk, lactose agar, glucose gelatine; its size and shape; its motility. The differences are so much within the limits of variations observable in that organism, that I am still uncertain whether the differences I have observed are enough to allow of its being considered a different species. It does not either correspond exactly to any of the bacilli resembling closely the bacillus coli. It is possibly only a pathogenic variety, but for the present it will be enough to say that it is *a bacillus of pathogenic milk.* In a further communication I will deal more in detail with this bacillus, and discuss the probable share which milk has in the production of human disease.

10. One of the outcomes of this investigation is that the ordinary methods of examination used for the detection of bad milk do not fulfil adequately their purpose.

An examination of the tables I have given in this communication will show that the Sp. gr., the reaction, the quantity of cream, the presence of cells or of microbes, taken separately or together, are insufficient indices of the pathogenic properties of milk.

An excessive number of cells is undoubtedly a very suspicious sign, especially when many of these cells are leucocytes. 'About one-half of the specimens very rich in cells were found to be capable of producing tuberculosis, but, tuberculous specimens excluded, there were some poor in cells which were exceedingly noxious. The number of microbes is a still less certain guide.

Unless I am greatly mistaken in my conclusions, it would seem that if cowsheds and the distribution of milk were under proper supervision, a very small amount of bad milk, if any at all, should reach the consumer. The chief disease to be dreaded, as communicable by milk, is tuberculosis, and there can be no doubt that usually it is only the milk of tuberculous cows with tuberculous udders which is capable of producing tuberculosis experimentally.[1] Surely such a danger could well be eliminated by general, systematic, and frequent inspection of *all* cowsheds by *competent* veterinary surgeons. The effects of this system of inspections should be periodically tested by investigations such as the one so successfully conducted in Manchester by Dr Niven, to whose valuable report I must refer the reader for much information not alluded to in this communication.

Work of this kind should be done under the direction of Public Health Authorities. Any other course would cause great loss of time and money. I am not, in saying this, overlooking the importance of the agricultural interests, but as the protection of human life is an object of paramount importance, I am convinced that more good will be obtained by harmonious joint action of the veterinary and medical professions than by independent action of either.[2]

As to the other sources of pathogenic properties, they could also be checked by proper inspection, having for object to obtain, (1) cleanliness in the byre, and of milk vessels. (2) Rapid transit of milk. (3) The keeping of the milk at as low a temperature as possible. I do not offer these as new suggestions, but as well recognised desiderata, put forth by many authorities, and which the present investigations show in a remarkably clear manner to be of great importance. I hope to be able to allude to the work of some of my predecessors on a future occasion.

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[1] It must be well understood that I do not mean to infer that the most common source of human tuberculosis is to be found in the ingestion of tuberculous milk. Undoubtedly in the great majority of cases infection takes place through the lungs. But because infection through the bowel is not the most common source of tuberculosis, this is not a reason why it should be neglected. It is easier to protect children against this danger than against inhalation of tuberculous dust, and it would be madness not to attempt to combat the lesser evil because there is a greater one more difficult to contend with. Some doubt regarding the value of experiments on guinea-pigs might be entertained. It may be justly argued that guinea-pigs are more liable to tuberculosis than man, and also that injection of tuberculous matter into the tissues is more likely to produce tuberculosis than ingestion of the same material. This is all very true, but it must not be forgotten that the difference between man and guinea-pigs, between subcutaneous inoculation and ingestion, are only matters of degree; they depend on the quantity of bacilli present. The same milk which is capable of producing tuberculosis in the guinea-pig will be capable of producing it in man wherever the number of bacilli is great. It must be remembered that young children are very susceptible to tuberculosis. Young animals, such as rabbits, guinea-pigs, cats, pigs, calves, can be easily rendered tuberculous by feeding on tuberculous milk.

To meet the objections that might be based on quantity, I have inoculated guinea-pigs with the sediment of 1 ounce and a half of milk. If that quantity of milk can produce tuberculosis in a guinea-pig weighing 10 ounces, it may be assumed as a possibility that 1 pint of the same milk would contain enough bacilli to produce, under conditions favourable to infection, tuberculosis in a child, not weighing more than 8 or 10 lbs. A single tuberculous cow yielding (as in one of the Manchester tuberculous cases) 36 pints of tuberculous milk, might produce enough milk in one day to infect a large number of children. Fortunately the conditions favouring infection do not occur frequently, and I speak here only of a potential danger. Yet if we take the case of an infant entirely fed on the milk of a single tuberculous cow it must be owned that the child runs considerable risks of becoming infected when the udder of the cow becomes tuberculous. Under similar conditions, kittens, young rabbits, pigs, and calves, would readily become infected.

[2] It might be urged that I attach too much importance to the lesions produced in the lower animals by inoculation of milk. I hope it will be well understood that I do not suggest that because a specimen of milk is capable of producing lesions in the guinea-pig, it would necessarily have caused disease in man. But, if I have proved that milk from healthy cows, and well kept, can be injected in large quantities without affecting the health of the animal, and, that milk of inferior quality is capable of producing various lesions, I am justified in saying that *milk causing these lesions is bad milk*, and that, whether it would cause disease in man, or not, *it is not right that it should be allowed in the market.* It is quite easy to obtain milk free from noxious properties, and why should one tolerate the existence of a public danger?

THE SERUM THERAPEUTICS OF TETANUS.[1]

By Professor NOCARD, Paris.

TREATMENT OF CONFIRMED TETANUS.

AT the end of the year 1895 I described to the Academy of Medicine some first results which I had obtained in the serum therapeutics of tetanus in the horse. When employed with the object of prevention the antitoxic serum was marvellously successful; when applied to the treatment of declared tetanus it almost always failed, even when the serum was injected in large quantity almost immediately after the appearance of the first symptoms of the disease. All the acute cases had a fatal termination; the recoveries were obtained only in cases of chronic tetanus exhibiting slow progress from the outset, and these, without doubt, would have recovered without the employment of serum.

This is because tetanus does not appear until several days after the absorption of the tetanic poison. During this long period of incubation the toxin absorbed has the whole time been exercising on the cells the action which is proper to it, and from which the tetanic symptoms proceed. Hence, if the dose of toxin absorbed is sufficient to entail death, nothing can prevent the effects of the toxic agent, which have already been silently effected for several days. The patients treated even by massive doses of serum die just like those which have not received any treatment.

In support of this interpretation I produced some experiments which seemed to show that it was well founded.

Some months ago the *Deuts. Med. Wochenschr*, 1896, No. 43, inserted a note announcing that the firm of Meister Lucius and Brüning, of Hochst-sur-Main, were prepared to supply medical men and veterinary surgeons with a serum prepared by MM. Behring and Knorr, by means of which it was possible to cure confirmed tetanus in man and in the horse. This serum is sold in a dry state, and the curative dose is 5 grammes. To employ it, it is necessary to dissolve the 5 grammes of dry serum in 45 grammes of sterilised water heated to 40°, and the solution ought to be injected as a single dose. For the horse it is better to inject the solution into the veins. The serum thus acts completely and rapidly, and one gains twenty-four hours as compared with the subcutaneous injection. In the case of the human subject also the intravenous method of injection is preferable. "If one makes the injection under the skin one cannot reckon on success in the acute cases unless one intervenes during the first thirty-six hours after the onset of tetanus. Hence, this remedy ought to be kept in stock in the large hospitals and veterinary institutes. The firm sells the curative dose at the price of 30 marks (30 shillings), which is a very moderate cost considering the efficacy of the product and the great expense of its manufacture" (*sic*).

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[1] **Translated from the "Recueil de Médecine Vétérinaire," 15th August 1897. (*Etudes expérimentales et cliniques.*)**

Since the above notice appeared in the *Deuts. Med. Wochenschr.* the German veterinary journals have reported a certain number of observations referring to cases of tetanus in the horse treated by the antitoxin of Höchst. The number of successes obtained have not amounted to the half of the cases treated; besides, we do not know all the cases of failure. I know, for example, that at the Veterinary Institute of Buda-Pesth three tetanic horses have been treated and all three have died. My colleague at Alfort, M. Cadiot, has not been more fortunate in two cases, where the disease did not appear to be exceptionally grave.

The results of practice thus do not seem to confirm the expectations of the *Deuts. Med. Wochenschrift.* I am well aware that the conditions of practice hardly justify one in forming an opinion in such a case. Most frequently the veterinary surgeon is consulted somewhat late, several days after the appearance of the first symptoms; the non-success of treatment may be ascribable to this delay in intervention. On the other hand, when the tetanic horse recovers after the injection of serum, who can say that it would not have recovered without it? In the one case, as in the other, one has only probabilities; certainty is lacking. All that an attentive study of the published observations justifies one in saying is that in promising success "when the injection is made during the thirty-six hours that follow the appearance of tetanus," the author of the note in the *Deuts. Med. Wochenschrift* is going much too far. To be convinced of this it suffices to read the observation which has been published by Professor Fröhner of Berlin ("Monatsh. für Thierh.," 1897, p. 298), which may be summed up as follows: A horse under treatment in his clinique was taken with slight trismus on the 29th January; three hours after the appearance of the first symptoms 5 grammes of the dry antitoxin of Höchst dissolved in 45 grammes of boiled water were injected into the jugular vein. On the 30th, the trismus had increased; a new injection of 5 grammes of antitoxin. On the 31st, state very serious, tetanus generalised; third injection of 5 grammes of antitoxin; death during the night.

In this case one cannot complain either of delay in intervention or of insufficiency of the dose of antitoxin injected. The animal received three times the "curative dose" of the dry serum, and the first injection was made three hours after the appearance of the first symptoms. In practice one can hardly hope for more favourable conditions, but nevertheless the patient succumbed.

Perhaps Fröhner had in this instance to deal with a case of exceptional gravity?

The truth is that clinical observation is inadequate to resolve the problem; one requires a considerable number of facts observed in identical conditions. Besides, one ought to take into account the country where the tetanus is developed, the nature of the traumatic injury which has provoked it, the duration of the incubation, etc.—all which conditions have a great influence on the gravity, progress, and termination of the disease.

In reality it is only by experimentation that one can prove the true value of a curative treatment of tetanus.

That is what I have attempted to do in a series of very simple experimemts, which have consisted essentially in injecting into several horses a dose of tetanus toxin sufficient to kill with certainty all the inoculated horses. Then, certain of these horses being kept as control animals, the others were treated according to the indications of the *Deuts. Med. Wochenschrift.*

The tetanus toxin which I have used is prepared by M. Marie, in the laboratory of M. Metchinkoff. It is in the state of a powder, and it is kept in a vacuum protected from light and heat. It has retained all its activity for more than a year. This toxin kills small mice in the dose of a ten-millionth of a milligramme; in this dose it gives tetanus to large mice, but it is necessary to go up to one-millionth of a milligramme in order to be sure of killing the whole of them. Guinea-pigs of 500 grammes die tetanic when they receive one-hundredth of a milligram of the same toxin.

In a first series of experiments, carried out on sixteen horses, I determined what was the minimum dose of this toxin certainly fatal when injected into a horse. For horses weighing from 430 to 480 kilogrammes the minimum invariably fatal dose was 6 milligrammes; horses which had received only 5 milligrammes resisted in the proportion of one out of three, and in one experiment one out of two. As a rule the first symptoms appeared after an incubation of six full days; in some subjects they showed themselves after five days, and in some others only after seven days of incubation. The duration of the disease varied from two to seven days; it was the Hungarian and Galician horses which took the disease soonest and died most quickly; the Norman and Danish horses are a little more resistant.

The minimum dose of toxin which is always fatal to the horse having been ascertained, it became easy to ascertain the exact curative value of the dry serum of Höchst. The only difficulty lay in the high price of this product; I have just mentioned that at the beginning the firm of Höchst demanded 30 marks (30 shillings) for each curative dose of dry serum, but when we wished to obtain a certain number of flasks of this serum we were asked to pay double the price (60 marks, or £3) of the curative dose of 5 grammes of dry serum. The expense exceeded the resources of my budget.

I have surmounted the difficulty by replacing, in the majority of my experiments, the serum of Höchst by an equivalent dose of the serum prepared at the Pasteur Institute; it was therefore necessary at the outset to ascertain the relative value of these two serums. M. Metchnikoff has been good enough to undertake this delicate operation. On various occasions he has carried out parallel experiments with a dry serum supplied by the firm of Hōchst on the 11th of December 1896, and a dry serum prepared at the Pasteur Institute in the month of February 1897, furnished by Horse No. 4. In all these experiments these two serums have shown an activity practically equal; in the dose of 0gr. 000,000,000,1 (one ten-billionth of a gramme) the two serums preserve the mouse against a dose of toxin ten times greater than the fatal dose, and 100 times greater than the tetanising dose. The serum of the Pasteur Institute proved itself a little more active than that of Höchst, for in the last experiments several mice treated by the serum of Hōchst had tetanus of the inoculated limb, while all those treated by the other serum remained unaffected.

The serum of the Pasteur Institute having an activity at least equal to that of the serum of Höchst, it was possible for me to multiply the experiments.[1]

Experiment I.—On the 14th March 1897, at 10 A.M., there were submitted to me four horses which had received on the 9th March preceding, under the skin of the neck, 6 milligrammes of dry toxin of the Pasteur Institute. One of the four, No. 60,738, was manifestly tetanic; in the morning at 7 o'clock it had been found with the tail elevated; it turned all in a piece. This horse was chosen as a control subject.

The three others (Nos. 52,824, 45,465, and 57,624) presented absolutely nothing abnormal at 7 A.M. when the stable was entered. At 10 o'clock I had them taken out in order to examine them at leisure; their gait was then a little stiff, and as I considered them as already affected, I injected them into the jugular; to the first 10 grammes of dry serum of the Pasteur Institute dissolved in tepid water, and representing 100 cc. of liquid serum, and to each of the two others 100 cc. of liquid serum.

On the 15th March the four animals were very ill; their legs were as stiff as posts, trismus very marked, appetite gone.

The control subject and Nos. 52,824 and 45,465 fell down on the night of the 16th in a condition of generalised tetanus; they died during that night.

In No. 57,624 the tetanus evolved less quickly; it was not generalised till the 17th March. The horse fell on the night of the 18th, and he was found dead on the morning of the 19th.

It will thus be seen that the intravenous injection of antitetanic serum practised on the appearance of the premonitory symptoms of tetanus is powerless to prevent the deadly progress of the disease, even when the dose injected is double that indicated as curative.

The results are identical when one injects the dry antitoxin of the firm of Höchst.

Experiment II.—On the 28th April at 4.30 P.M. I injected 6 milligrammes of dry toxin under the skin of the neck of three Danish horses, Nos. 43,480, 48,993, and 52,663.

On the 4th May at 8 A.M. none of the three horses appeared affected; they were taken out in order to judge better; they were lively, vigorous, and supple in all their movements.

They were taken out again at 2 P.M., but nothing abnormal was observable.

At 5 P.M. they were taken out for the third time. No. 48,993 appeared a little stiff in the neck and loins, and its eyes were bright and prominent. When tested by pricking it was seen that the animal was affected; exactly six times twenty-four hours had elapsed since it was inoculated. I immediately injected into its jugular 5 grammes of dry serum obtained from Höchst (of the 11th December 1896).

The two other horses when carefully examined did not yet show anything abnormal.

On the 5th May at 6 A.M. the two untreated horses did not present any signs of tetanus. At 10 A.M. they were again examined, and then one observed that No. 43,480 was a little stiff in its movements. I immediately injected into its jugular 5 grammes of dry serum of Höchst (of the 11th December 1896), dissolved in forty-five grammes of sterilised water.

On the 6th May, No. 52,663, *which had been left as a control*, was manifestly affected.

On the 7th May the three horses had generalised tetanus: abdomen contracted, neck arched; they walked as if on posts; they still, however, ate a little.

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[1] These experiments have been carried out on thirty-six cast horses of the Compagnie Générale de Voitures; these horses have been graciously placed at my disposal by M. Bixio, to whom I here express my thanks.

On the 8th and 9th all the symptoms were aggravated. The jaws could not be separated, absolutely impossible to take food.

On the 10th May the three horses were still standing, but in a miserable condition, fixed with their legs spread out and rigid as posts; all the muscles contracted, the nostrils dilated to an extreme degree, the neck violently twisted, and the head extended on it.

At 5 o'clock No. 43,480 (one of the treated) fell down with its legs as stiff as bars of iron and with a rattling in its throat; it died during the night.

No. 52,663 (control) fell and died on the 12th May.

The last died during the night of the 12th to the 13th.

One does not obtain any better results even when the injection of antitoxic serum is practised twenty-four hours before the appearance of the first symptoms.

Experiment III.—On the 9th March, at 10.30, I injected under the skin of the neck 6 milligrammes of dry toxin to five horses, Nos. 42,153, 45,550, 49,645, 54,407, and 55,718.

On the 14th March, at 10 o'clock, none of these horses, when examined individually and very closely, presented the least symptoms suggestive of tetanus. Into the three first I injected by the jugular 100 cc. of antitetanic serum from the Institute Pasteur (furnished on the 15th February, by horse No. 4); the two last horses were retained as control subjects.

On the morning of the 15th March the five horses presented some suspicious symptoms; in the stable one did not notice more than a little elevation of the tail; it was necessary to lead them out in order to discern a little stiffness of the neck and legs. On the 16th the tetanus was very decided in all the animals, but the whole of them were still eating. On the 17th generalised contraction, tail elevated, neck incurved, nostrils dilated, the prehension of food almost impossible. On the 18th the state of all the horses was still more aggravated. About 10 A.M. one of those treated (No. 42,153) fell down, rattling in its throat and struggling; it died the same night. One of the control animals (No. 54,407) died on the 19th at 4 A.M., the other about 5 P.M. The two other treated horses fell down on the 20th, and died during the night.

It will thus be seen that the intravenous injection of a considerable dose of antitoxic serum is powerless to prevent the evolution of tetanus provoked by a dose of toxin always fatal for the control subjects, even when this injection is practised twenty-four hours before the appearance of the first symptoms.

If one intervenes forty-eight hours before one has much more chances of saving the patient; in some cases one even prevents the appearance of any symptom of tetanus, but it is necessary to inject large doses of serum, and to make the injection into the veins.

Experiment IV.—(1) On the 24th February 1897, I injected 6 milligrammes of dry toxin under the skin of the neck of horses No. 46,214 and 49,344.

On the 28th February I injected into the jugular of the first 100 cc. of serum from the Pasteur Institute (furnished on the 15th February by the immunised horse No. 4).

The other inoculated horse was retained as a control subject.

On the 2nd of March, at midday, the control subject was manifestly affected; its tail was elevated, it was stiff in the neck and loins. On the 3rd March tetanus generalised, abdomen retracted, legs stiff as posts, nostrils extremely dilated, head extended on the contracted neck. It died during the night.

The horse treated never presented the slightest symptom of tetanus.

(2) On the 9th March a Danish horse (No. 54,096) received under the skin, at the same time as other thirteen horses, 6 milligrammes of toxin.

On the 13th I injected *under its skin* 60 grammes of serum from the Pasteur Institute (of the 15th February, from the immunised horse No. 4). Two other Danish horses (Nos. 54,407 and 55,178) left as control subjects were not seized till the 15th March; the treatment thus preceded by forty-eight hours the onset of tetanus. Nevertheless, on the 16th the animal treated was clearly tetanic; on the 18th tetanus was generalised; death supervened on the 21st March, two days after that of the control subjects.

The dose of serum injected would, without doubt, have been sufficient to save the animal if the injection had been made into the jugular in place of under the skin.

A last series of experiments well shows the high preventive value of antitoxic serum: a very feeble dose of serum suffices to prevent the appearance of tetanus when the injection is made even under the skin a little time after the animal has received the dose of toxin which is always fatal for the control subject.

Experiment V.—On the 9th March, at 10 A.M., I injected under the skin of the neck of fourteen horses 6 milligrammes of toxin. Of these horses three not treated and serving as control subjects became tetanic, one (Hungarian) on the 14th March, the two others (Danish) on the 15th March. Seven others treated either the moment the first symptoms appeared, or twenty-four to forty-eight hours previously, all contracted fatal tetanus.

The four last resisted, thanks to a less tardy treatment.

The horse No. 46,443 (Normandy) received under the skin 20 cc. of serum of the Institute Pasteur one hour after the inoculation of the toxin; it never presented the least symptom of tetanus.

The horse No. 49,298 (Normandy) received under the skin 30 cc. of the same serum twenty-four hours after the injection of toxin; that also never appeared to be affected.

The horse No. 54,378 (Danish) received under the skin 30 cc. of serum forty-eight hours after the toxin; a little stiffness and incurvation of the neck showed themselves on the 19th March, and persisted during fifteen days, but the animal retained its appetite, spirit, and suppleness of its members. It never appeared in serious danger; complete recovery rapidly supervened.

The horse No. 60,277 (Hungarian) received under the skin on the 12th March (three full days after the injection of the toxin), 40 cc. of serum. On the morning of the 14th March it was manifestly tetanic; the same day I injected into its jugular 75 cc. of serum. Up to the 25th March the disease steadily became worse, and then the symptoms gradually improved to such a degree that on the 4th April the animal was almost quite recovered, save for a slight incurvation of the neck on the side on which the toxin had been injected.

If one considers the extreme severity of this experiment,—the dose of toxin, invariably fatal for the control subjects, having been injected all at one time, and immediately absorbed,—one may affirm that in practice one would have a much better chance of delaying the development of tetanus, or preventing its appearance, by intervening several hours after the infliction of the wound. But it is very necessary to remember that the longer the period that elapses before intervention, the larger ought to be the dose of serum injected.

From this long experimental study I believe I am justified in concluding, as I did in 1895, that the curative treatment of tetanus has still to be discovered.

That is not to say that one ought to renounce the employment of antitoxic serum in the treatment of confirmed tetanus.

On the contrary, it is my profound conviction that the injection of serum is the best method of treatment to which one can have recourse. As a matter of fact, all my correspondents who have treated tetanic horses by the injection of serum declare themselves very well satisfied, not that the number of recoveries obtained has been higher, but the crises have been less numerous and less intense, and when recovery has supervened convalescence has been more rapid.

On the other hand, it frequently happens that the traumatism from which the tetanus proceeds escapes the closest examination ; one cannot then practise "eradication of the focus," so justly recommended by M. Berger. The elaboration of the toxin is then continued, thus incessantly augmenting the tetanic intoxication ; it is especially in these cases that the serum will prove useful ; although powerless against the toxins already absorbed at the moment when treatment is begun, it will be able to destroy as fast as they are formed the toxins which will be produced in the undiscovered focus.

Unhappily these are the rarest cases, and more frequently the employment of antitoxic serum does not prevent the tetanus from effecting its evolution, which is so frequently fatal. Although it may thus be necessary to continue the employment of the serum in the treatment of declared tetanus, he who counts on curing the majority of his patients will expose himself to a cruel disappointment. To-day, as yesterday, the most elementary prudence dictates the preventive injection of serum in every sort of injury which we know to be frequently followed by tetanus.

The experience of veterinary surgeons shows the excellence of this method. The results already obtained in 1895 entirely confirm those which I have already published.

II.—PREVENTIVE TREATMENT.

In the foregoing I have narrated experiments proving that a very small dose of antitetanic serum suffices to prevent the appearance of tetanus in the horse, when the injection is made a little after the animal has received a dose of toxin that is always fatal to the control subjects. On this main point the result of the practice of veterinary surgeons entirely confirms the experimental results.

From the 1st August 1895 to the 1st June 1897, I have distributed to veterinary surgeons, French for the most part, nearly 7000 flasks containing 10 cc. each of antitetanic serum. The preventive treatment comprises two injections of 10 cc. each, and hence the serum which I have sent out ought to suffice to confer immunity against tetanus on about 3500 animals.

Thanks to the kindness of the majority of my correspondents, I know the results of treatment for 2705 subjects, of which 2373 were horses or mules, 44 bulls, 82 rams or lambs, and 206 pigs. Each of these animals received two injections of serum,[1] with an interval of ten to twelve days, being 20 cc. for the large animals, and 6 to 10 cc. for sheep and pigs. The details will be found in the accompanying tables (*see* page 216).

These 2705 animals may be divided into two very distinct groups.

The first group, which is much the more important, comprises over 2300 animals which received the first injection of serum immediately after the operation which they had undergone, such as castration, amputation of the tail, excision of scirrhous cords or tumours, operations on cryptorchids or for umbilical or inguinal hernia, etc. *Not a single one of these 2300 animals has taken tetanus.*

The second group is much less numerous, though it comprises nearly 400 subjects; these did not receive the first injection until one, two, three, four days, *or more* after the accidental injury of which they had been the victims, such as pricks from gathered nails, pricks in shoeing, quittor, wounds from harrow teeth, kicks, bad falls, wounds soiled by earth or dung, etc.

In the case of the animals of this group one would, *a priori*, expect the results of treatment to be less satisfactory than in animals of the first group, for one would fear that in a certain number of the subjects the injection of serum would be carried out too late to prevent the evolution of tetanus.

These fears have not been realised. *None of these 400 animals treated at a longer or shorter interval after the injury has succumbed to tetanus.*

Only one horse, *treated five days after the accident* (*prick in shoeing*), presented tetanic symptoms. The disease was quite benign; it appeared on the 10th December and it disappeared on the 22nd December. The animal never ceased to eat, and it never appeared to be in danger. The serum had been injected too late and in too feeble a dose to prevent the appearance of tetanus, but it had at least considerably attenuated the gravity of the disease, since the animal was able to return to work twelve days after the onset of the attack.

To comprehend the importance of the results obtained, it will suffice to glance at the accompanying tables. One will there see, for example, that all the animals treated belong to stables, farms, or villages where tetanus had claimed victims some days, weeks, or months previously. A number of them were the immediate neighbours of animals suffering from tetanus.

In certain cases the traumatic injury had been produced at the same time and in the same conditions as in other animals untreated, some of which became tetanic.

In a number of cases tetanus was so common in the locality that the owners had given up submitting their animals to certain operations which are usually performed in order to augment their market value, such as castration, docking, operation for hernia, etc.

The majority of my correspondents had not had recourse to preventive treatment except where they had previously lost one or several animals after operation, However, almost all of them have had to register cases of tetanus supervening on animals in their practice which had not been submitted to an injection of serum. Thus, during the whole period covered by the observations, my sixty-three correspondents, who had not lost a single case out of 2727 animals treated preventively, have observed 259 cases of tetanus in animals not treated, of which 191 were horses, 57 rams, and 11 bovine animals.

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[1] **Several veterinary surgeons have made only one injection of from 10 to 20 cc., and the result has been no less good. I believe, however, that it is better to make two injections, on account of the short immunity conferred by the serum.**

I do not wish to weary the Academy with details of the facts observed, but I will ask permission to cite some of the more important of them.

I. Between the 1st January and the 4th April 1896, M. Bigot, veterinary surgeon at Bonneville, castrated without accident thirty-two horses; on the 4th April he admitted into his infirmary a tetanic horse from a farm where tetanus was so frequent that one could not castrate lambs there by any method involving the use of the knife without the majority dying from tetanus. On the 6th April M. Bigot castrated eight horses in a courtyard in his infirmary where the animal suffering from tetanus had been kept; between the eleventh and thirtieth day after the operation five of these eight horses died from tetanus. On the appearance of the first case M. Bigot requested me to supply him with serum, and he made some injections to all the horses which he had castrated since the 6th April, and which might have been contaminated like the first lot. These horses were to the number of twenty-eight; ten of them were castrated between the 12th and 15th April, and two of these took tetanus and died in spite of large doses of serum; not one of the other twenty-six horses, castrated on the 12th, 15th, 19th, 22nd, and 25th April, has become tetanic. Nevertheless, these had been contaminated like the others, for a horse in the same infected courtyard which was fired by pyro-puncture on the 10th May, and for which the employment of serum was not thought necessary, took tetanus and died there. Since then M. Bigot has castrated 163 horses without losing a single one of them, thanks to the preventive treatment with serum, although the majority of these belonged to farms where tetanus had claimed one or several victims in 1895 or 1896.

II. I have had occasion to point out to the Academy the frequency of tetanus in the north-west suburbs of Paris. One of my old fellow-students, M. Coret, in practice at Aubervilliers, has annually seen there for the last fifteen years from thirty to forty cases of tetanus in the horse.

Between the 1st August 1895 and the 1st June 1897, M. Coret submitted to preventive injections of antitoxic serum 116 horses, suffering from gathered nails, pricks in shoeing, wounds on the legs, etc., all of which wounds are particularly dangerous in regard to tetanus. Only one of these animals became tetanic, and this had been treated five days after the injury (prick in shoeing). The tetanus, however, was very benign; it lasted only for twelve days, and the animal has since done excellent work. In two years M. Coret has not lost a single one out of 116 horses which he has treated preventively. But during the same time he has been called to visit fifteen horses attacked with tetanus in consequence of analogous injuries, but not submitted to preventive injection. But for the employment of serum, M. Coret would have had at least fifty-five or sixty more cases of tetanus in the horse ; the difference appears to be a good measure of the benefits which are derived from the new method : thanks to it, he has saved at least forty horses in his practice from tetanus.

III. M. Pierrot of Stenay lost annually from ten to twelve animals from tetanus ; in 1896 he lost only three ; one of these was a horse castrated at the same time as four others, of which only three received injections of serum, M. Pierrot having exhausted his stock. Of the two horses not injected one took fatal tetanus. Tetanus is much rarer in cattle than in horses. M. Pierrot castrated on the same day one colt and two young bulls ; the colt only received an injection of serum ; one of the young bulls which were not injected died of tetanus.

IV. M. Tonnelier of Sièges observed an identical fact. Six colts were castrated on the same day in the same village. He could only make an injection of serum in the case of four of them ; one of the others took tetanus.

V. M. Mongin of Mirecourt records a fact of a different kind. Of five colts castrated at the same time one took tetanus ; the four others were immediately injected, and they remained healthy.

VI. Another analogous fact, proving the possibility of quickly putting a stop to the mortality in a flock where tetanus appears after castration.

On the 15th April M. Auvray castrated forty-five lambs ; on the 23rd April three died of tetanus. On the 24th the forty-two survivors received an injection each ; only one of these died from tetanus.

VII. In a piggery on the estate of d'Atour one could not castrate the young pigs without losing from 10 to 12 per cent. of them from tetanus ; since the employment of serum 206 have been castrated without a single case of tetanus.

I might multiply these examples, but I will confine myself to pointing out that a large number of my correspondents now never perform operations involving the use of the knife without completing them by a preventive injection of serum, and I will conclude by repeating, with M. Warnesson of Versailles, that "*when employed as a preventative the antitetanic serum is absolutely efficacious.*"

[TABLE.

PREVENTIVE INJECTIONS OF ANTITETANIC SERUM.

RESULTS OBTAINED FROM THE 1ST AUGUST 1895 TO 1ST JUNE 1897.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Auvray, St. Amand-de-Vendôme	3 Horses Castration 41 Lambs Castration	4 Lambs	Castrated on the same day forty-five lambs; on the eighth day three of them were seized with fatal tetanus. The other lambs were then injected; a fourth lamb died on the fourth day; the remaining forty-one continued in good health. A horse castrated on the same farm received a preventive injection and remained well.
Baillon, Oulchy-le-Château	2 Horses Castration	4 Lambs	On a farm where two other horses had just died from tetanus. Four or five cases of tetanus seen annually.
Barbry, Armentières	8 Horses Castration	1 Horse	In a commune where he had just lost a horse recently castrated.
Barry, Maximeux	17 Horses Castration	1 Horse	Had just lost a castrated colt; lost some every year. It was this that decided him to have recourse to serum.
Bégné, Saint-Servan	15 Horses Castration	4 Horses	Saw seven or eight cases of tetanus every year; has seen four cases in 1896, one of which had been castrated by him and not injected, owing to his having no serum.
Bernaud, La Gorgue	47 Horses or Mules 15 Castrations 29 Dockings 3 Gathered nails, or wounds of various kinds	3 Horses 2 Mules	All these animals belonged to farms where tetanus had previously prevailed. Tetanus is frequent in the district; M. Bernaud had seen in 1896 five cases in animals docked and not treated.
Berque, 6th Chasseurs, Mascara	20 Horses Castration	6 Horses	Tetanus frequent in the district; in 1896 had seen six cases of tetanus in the horse after castration by the natives.
Bertrand, Maurage (Belgium)	5 Horses Castration and Docking	1 Horse	These five horses belonged to a farm where two horses had died from tetanus during the preceding year. M. Bertrand had under his care at the same time a case of tetanus in the horse in the same village.
Bigot, Bonneval	189 Horses Castration	10 Horses	On the 4th April 1896, M. Bigot received into his infirmary a horse suffering from tetanus; on the 6th April he castrated in his courtyard, where the case of tetanus had been kept, eight horses, and five of these were seized with tetanus from the twelfth to the thirtieth day after castration. On the 22nd April all the horses castrated since the sixth received an injection of serum; the number thus treated was twenty-eight. Two which had been castrated on the 12th and 15th April were seized and died in spite of large doses of serum; the twenty-six others remained healthy. It was certainly the serum that preserved them, for a horse which was cast in the same yard on the 10th May, in order to be fired by pyro-puncture, was not injected, and it contracted fatal tetanus. Since then M. Bigot makes an injection after every operation, and none of the animals operated on by him has taken tetanus, but he has seen two fatal cases of tetanus in horses with wounds not treated with serum.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Biot, Pont-sur-Yonne	39 Horses Castration	1 Horse	M. Biot saw annually four or five cases of tetanus after castration. This year he castrated on the same day two colts on two different farms; only one of these received an injection of serum, and the other took fatal tetanus.
Bissauge, Orléans	2 Horses 1 Gathered nail 1 Serious wound	2 Horses	In two stables where two horses had just died from tetanus.
Bouchet, Creil	6 Horses 2 Gathered nails 1 Docking 3 Serious wounds	2 Horses	The two horses with gathered nails were neighbours to two cases of tetanus.
Bouquet, Chablis	8 Horses 5 Castrations 1 Gathered nail 2 Serious wounds	1 Horse 6 Lambs	These eight horses belonged to villages where tetanus had previously prevailed. Had seen seven cases of tetanus in 1893; one horse and six lambs not injected.
Carrière, Saint-Affrique	32 Horses 25 Castrations 5 Scirrhous cords 2 Cryptorchids	6 Horses	Had seen in 1896 six cases of tetanus in horses that had been castrated without preventive injection.
Cavel, Guise	60 Horses Castration Docking	1 Horse 15 Lambs	In communes where he lost each year some horses after castration or docking; does not now operate without injecting serum as a preventative. In 1896 saw one case of tetanus in a horse with a wound, and fifteen cases in lambs castrated by a shepherd.
Coret, Aubervilliers	116 Horses 78 Gathered nails 2 Docking 2 Castrations 34 Operations or wounds of various kinds	15 Horses	The district of Aubervilliers is particularly bad for tetanus. During fifteen years M. Coret saw at least thirty cases of tetanus in the horse annually; in nearly two years he has seen only fifteen, and these were in horses not treated; the difference gives the measure of the benefits which he has derived from the treatment. One of the horses presented very distinct symptoms of tetanus some days after the injection, but this animal had been treated five days after the injury (pricked foot), and had received only 10 cc. of serum; besides, the disease was very benign; it appeared on the 10th December, and completely disappeared on the 22nd December.
Crouzel, Coutances	4 Horses 2 Castrations 2 Serious wounds	—	In a district where tetanus is very frequent in man and animals.
Declaude, Troyes	9 Horses 6 Castrations 3 Cryptorchids	—	—
Denis, Genappe (Belgium)	53 Horses 36 Dockings 15 Castrations 2 Gathered nails	7 Horses	In a district where tetanus is very frequent. At the same time M. Denis saw seven cases of tetanus in horses which had not been treated.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Didier, Dun-sur-Meuse	7 Horses Castrations	4 Horses	In villages where tetanus had been observed in preceding years; in 1896 M. Didier saw four cases of tetanus in horses that had not been treated.
Dieudonné, Einville	44 Horses Castrations Docking Umbilical hernia Serious wounds	11 Horses 3 Oxen 2 Sheep	In farms or villages where tetanus is frequent in man and animals.
Douarche, Maule	11 Horses Castrations Gathered nails Wounds	2 Horses	Tetanus frequent in the district.
Dubois, Landivisiau	54 Horses Castrations	(?)	Lost several horses every year after castration. Now does not castrate without having recourse to preventive injections.
Dupuy, 8th Artillery, Nancy	3 Horses 1 Gathered nail 1 Castration 1 Multiple wounds	1 Horse	Had for a patient at the same time one horse affected with tetanus.
École de Saumur	27 Horses 23 Castrations 1 Docking 3 Wounds of various kinds	4 Horses	At the same time four horses not treated were seized with fatal tetanus.
Feutry, Domaine d'Autour, Belgium	206 Young Pigs or Sows Castration	(?)	Lost each year over 10 per cent. of the pigs castrated. Does not now castrate without injecting 3 to 5 cc. of serum; no further loss.
Fouque, Marseille	47 Horses Castration Gathered nails Docking 12 African Rams Castration	5 Horses 18 Rams	Saw at the same time five cases of tetanus in horses not injected. Out of forty African rams previously castrated in the same locality by the same means eighteen were seized with tetanus almost at the same time; the twenty-two others were immediately sold to the butcher. Twelve rams were castrated one month afterwards without accident, thanks to a preventive injection of 5 cc. of serum.
Gabriel, Ribemont	6 Horses Docking	2 Horses	On two farms where one horse had just died from tetanus. Tetanus was so frequent in the district, especially after docking, that the majority of owners had given up that operation.
Guénard, Nonancourt	14 Horses Castration	2 Horses	On farms or in villages where tetanus had been observed during the previous year. In 1896 had to treat two cases in horses not injected.
Henry, Tunis	7 Horses Castration	1 Horse	Had to treat at the same time one horse attacked with tetanus after wounds on the neck.
Huet, Eucaussines-d'Enghien (Belgium)	6 Horses Castration Docking	1 Horse	On farms where one or two horses had been lost every year from tetanus. The farmers would no longer venture to have their horses castrated or docked. On one of them a horse had died from tetanus a month previous, after a wound of the coronet.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Jacquot, Saint-Nicolas-du-Port	15 Horses 5 Castrations 2 Gathered nails 4 Dockings 2 Umbilical hernia 2 Serious wounds	2 Horses	In villages where tetanus had formerly prevailed. Had seen two cases in 1896, in horses not injected.
Jouquan, Vitré	5 Horses 2 Castrations 1 Umbilical hernia 2 Dirty wounds	7 Horses	Tetanus very frequent in the district, especially after operation for hernia by empirics; had seen seven cases in six months.
Lapérierre, Vervins	146 Horses Castrations Dockings	(?)	In villages where tetanus had formerly prevailed. "*Preventive injection of serum is an indispensable accompaniment of castration and docking.*"—Letter of 4th April 1897.
Le Morvan, Rouen	15 Horses 10 Castrations 4 Nickings 1 Gathered nail	1 Horse	The case of gathered nail was a circus horse, companion of another dead of tetanus a short time previously.
Loix, Nivelles (Belgium)	225 Horses 125 Castrations 20 Dockings 15 Umbilical hernia 65 Gathered nails, pricks, and dirty wounds	2 Horses	In stables or villages where tetanus had formerly prevailed; saw six or seven cases of tetanus every year; had not seen any cases in 1896, and one case in 1897. "*No longer operates without at the same time making an injection of serum.*"
Lorin, Meaux	7 Horses Castrations, gathered nails, and various wounds	1 Horse	In a stud where several cases of tetanus had been produced, of which one case occurred in 1896.
Marange, Vézelise	6 Horses 5 Castrations 1 Wound from scythe	1 Horse	The castrated horses belonged to three farms, two of which had cases of tetanus in the preceding year. The animal with a wound occupied a stable where six weeks previously a horse had died from tetanus after castration. M. Marange saw every year five or six cases of tetanus.
Marion, Ravières	23 Horses 19 Castrations 2 Dockings 1 Umbilical hernia 1 Gathered nail	1 Horse	In communes where tetanus had formerly prevailed.
Mathieu, Attigny	171 Horses Castration and Docking 30 Bulls and 29 Rams Castration	3 Horses 1 Man	Tetanus frequent in the district, both in man and in animals.
Mercier, Fontenay-le-Comte	17 Colts and young mules 6 Castrations 10 Umbilical hernia 1 Dirty wound	6 Colts and young Mules	In villages where tetanus is very frequent; saw six cases this year in subjects not injected. One of the foals treated was born in a stable where two other foals, which were not treated, died from umbilical tetanus, one of them only a few days previously.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Mesnard, Hiersac	30 Horses 5 Asses 2 Various wounds 33 Castrations	4 Horses and Asses	The majority of them belonged to stables or villages where tetanus had prevailed the year preceding.
Mollereau, Charenton	29 Horses 21 Gathered nails 8 Dirty wounds	5 Horses	Eleven of these horses belonged to an establishment where two horses with insignificant wounds and not injected took tetanus; all these animals had for litter dirty straw which had been used for the packing of mineral water bottles carried by boat. Had also observed three other cases of tetanus in horses not injected.
Mongin, Mirecourt	4 Colts Docking	1 Colt	Out of five colts docked at the same time one took tetanus; the other four were injected immediately and remained healthy.
Monod, Fort-de-France (Martinique)	8 Horses 1 Mule 6 Gathered nails 1 Docking 2 Dirty wounds	1 Ram	In studs where tetanus was very frequent both in man and animals. At Martinique the losses from tetanus are considerable; many owners do not dare to have their horses castrated or docked.
Mourot, Tunis	4 Horses 3 Castrations 1 Suppurating sore	——	Had two cases of tetanus in the horse in 1895.
Moreau, Sézanne	4 Horses (?)	1 Horse	Were companions to a fifth horse dead of tetanus.
Mulotte, Chateau-Salins	46 Horses Castrations and serious wounds	15 Horses 1 Bull	These forty-six horses belonged to thirty-two stables, fourteen of which were infected; the others were adjacent to these. M. Mulotte no longer treats any sort of traumatic injury without making an injection.
Pavot, Vendegies-au-Bois	38 Horses 22 Castrations 8 Dockings 8 Various wounds and umbilical hernia	(?)	In farms or villages where tetanus had been previously observed.
Picard, Sourdon	34 Horses 15 Castrations 19 Dockings	3 Horses	Saw at the same time three cases of tetanus in the horse.
Pierrot, Stenay	140 Horses 132 Castrations 8 Gathered nails and wounds	2 Horses 1 Bull	Saw each year ten to twelve cases of tetanus; has only seen three this year: namely, one horse operated on for hernia and not treated; the second castrated the same time as four others, three of which could not be injected owing to want of serum. The young bull was castrated at the same time as a colt which was injected, and only the young bull was seized with tetanus.
Plessis, Blois.	24 Horses Castration, Docking	1 Horse	Has not lost any horses since he makes injections after all his operations. Had seen in 1896 one case of tetanus in a horse not injected.
Quentin, Tilly-sur-Meuse	7 Horses Castration	1 Child	In farms where tetanus had previously been observed; on one of them a child had just died.

Veterinary Surgeons Operating.	*Animals Treated. Nature of Injury.*	*Cases of Tetanus observed among the animals not Treated.*	*Observations.*
Ravier, Saint-Mihiel	135 Horses Castration and Docking 14 Bulls Castration	2 Horses 2 Cattle 2 Men	On farms where tetanus had been observed previously both in man and in animals. In 1896 saw fatal cases in a man, a young girl, two horses, and three cattle. No longer operates without preventive injection.
Saudé, Buzancy	184 Horses 175 Castrations 3 Scirrhous cords 5 Umbilical hernia 1 Gathered nail	12 Horses 11 Rams	Tetanus very frequent in the whole district, especially in colts, young bulls, and rams operated on by castrators.
Simon, Lunéville	6 Horses 2 Castrations 3 Dockings 1 Umbilical hernia	(?)	In a village where, out of three colts castrated last year, two took tetanus.
Tayot, Launois	40 Horses Castrations, Docking, hernia, and various wounds	7 Horses 3 Calves	Tetanus very frequent in the whole district. Saw at the same time seven horses and three calves that had not been injected affected with tetanus.
Tonnelier, Les Sièges	13 Horses 12 Castrations 1 Umbilical hernia	2 Horses	In a commune where six horses were castrated the same day, and only four of them injected owing to want of serum; one of the two others took fatal tetanus.
Treuvelot, Manouba (Tunis)	(No figures given)	(?)	"Had formerly a great deal of tetanus; no longer has cases after his operations since he injects serum as a preventative."
Vernaux, Rouen	65 Horses 62 Castrations 1 Hernia 1 Scirrhous cord 1 Docking	—	Formerly saw two or three cases of tetanus every year. One of his castrated horses showed on the 12th day a little stiffness of the neck and legs; this all disappeared in some days; the animal had only received one injection of 10 cc.
Villedieu, Die	(Does not give any figures)	8 Colts	In a village where he had for a patient a donkey suffering from tetanus, he castrated on the 5th March five horses, one of which died on the 1st April. He injected serum to the four others, one of which already showed a little trismus; that one died on the 7th April, and the other three remained well. Tetanus is frequent in the district; last year out of eight colts castrated on the same day six died. Mules recover in the proportion of 60 per cent.
Warnesson, Versailles	64 Horses Serious accidental and surgical wounds	8 Horses	In localities where tetanus had previously prevailed. "*One may say that when employed as a preventative the serum is absolutely efficacious.*"
X. (signature illegible)	9 Horses 5 Docking 1 Wound 2 Castrations 1 Umbilical hernia	(?)	In farms or villages where he had previously lost horses from tetanus.

THE PREVENTIVE MEASURES DIRECTED AGAINST BLACK-QUARTER, AND AN INVESTIGATION INTO THE EFFICACY OF SETONING.[1]

By STEWART STOCKMAN, Professor of Pathology, Royal (Dick) Veterinary College, Edinburgh.

AT the present day it is known to all of us that black-quarter is a bacterial disease, and most of us, I think, believe that in the majority of cases it is contracted by inoculation of a wound in some part of the body. For the discovery of the bacillus of black-quarter, or charbon symptomatique, as it is called in France, we are indebted to Arloing, Cornevin, and Thomas. In 1880 they described and demonstrated the pathogenic properties of this bacillus, which they named the bacterium Chauvœi. It is true that in 1860 Feser described bacilli in the juice from black-quarter muscle, but Arloing, Cornevin, and Thomas were the first to give a proper account of the micro-organism. Since then they have furnished us with the greater part of our information regarding the pathology of this disease, and they have put into practice a method of preventive inoculation which has obtained great favour among continental veterinarians.

Their method of preventive inoculation, however, is not the only one in use. Among those who have done important work on this subject are Professor Kitt of Munich, and Dr Roux of Paris. These methods of prevention have not gained favour in the United Kingdom. It is only fair, however, to say that for good reasons they have not been extensively tried in this country. These reasons are—firstly, that the results of the first experiments were disastrous; secondly, that the wholesale inoculation of cattle against black-quarter is unwarrantable, because the disease is only known on certain farms, and even the warmest advocates of the present methods have to number some deaths from inoculation in their statistics. In this country one of the oldest and most popular of preventive measures directed against the disease is setoning, and some investigations which I have made into the efficacy of that measure will constitute a considerable part of this paper.

I shall discuss shortly each preventive method in use, but, before doing so, it will be as well to say a few words about the distribution of the disease, the virulent material, the susceptibility of animals, and the manner whereby they contract it, because a knowledge of these subjects is necessary before attempting to apply preventive measures.

I would like to explain here that our warfare is not accomplished when we have found even a good method of preventive inoculation. We should regard that method rather as a barrier to stem the tide, while we are applying more radical measures which take a longer time to operate.

Distribution of Black-quarter.—The disease in this country is confined to certain districts, and often in these districts to certain farms.

Veterinarians in different parts of this country will point out to you black-quarter farms with neighbouring establishments which are practically free from the disease. In some parts it would be well worth an intending tenant's while to inquire into the reputation of the farm in this respect. Strebel, in his valuable communication to the Sixth International Veterinary Congress, says that the soils in black-quarter districts have a common character. They are of a clayey nature, hence impervious and damp. This, of course, simply means that filth, including disease germs, is not drained away from such a soil, and therefore the plague is kept up for a long time. It applies to the germs of other diseases as well as that of black-quarter. Hess, after inquiring into the distribution of the disease in various parts, could not admit that any one kind of soil was particularly favourable. We know, however, that one of the best preventive measures is to drain the land, but unfortunately that is not always practicable.

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[1] **Read before the Midland Counties Veterinary Association at Birmingham on the 27th August, 1897.**

It does not come within the scope of this paper to describe all the characters of the black-quarter bacillus, but I should like just to remind you here that it is a sporulating microbe, an obligatory anærobe, and that its tendencies seem to be decidedly saprophytic. On the surface of the soil the bacilli, and probably for that matter the spores as well, do not remain active for any very long time. The combined effect of oxygen and sunlight is powerfully destructive.

They sink into the ground, however, and there is evidence to show that the spores at least, if not the bacilli, may here remain active for years.

With the earth taken from the damp parts of black-quarter pastures at a depth of 12 inches, Feser succeeded in inoculating sheep with black-quarter. The earth from the dry parts did not contain the pathogenic agent. Feser's examinations seemed to show that the bacillus itself is present in the soil, but of this more evidence is required. There are two possible explanations of how these sub-soil germs are brought to the surface again. The one is that it is connected with the rise and fall of the ground water (so-called), the other is that, when the ground cracks, as it does in hot weather, the dry sub-surface earth gets blown or scraped to the top. These explanations savour somewhat of the hypothetical, but I think they are worthy of further investigation.

Animals attacked.—The animals attacked by black-quarter are chiefly cattle and sheep. The goat is also susceptible, but the horse enjoys an almost complete immunity. Of the animals commonly used for experiment in the laboratory, the guinea-pig is the best reagent. The rabbit has a certain amount of immunity, but this can be overcome by injecting along with the virus such agents as lactic or acetic acids, certain other microbes, or the toxines of the black-quarter bacillus itself.

The micrococcus prodigiosus overcomes the immunity in the rabbit, but seems to strengthen the resistance of the guinea-pig. Bruising of the tissues at the seat of inoculation seems also to intensify the action of the virus. These agencies are believed to act by taking up the attention of the white cells and the other resisting forces of the animal organism.

It is interesting to know that certain agents increase the virulence of the black-quarter microbe, or rather let us say diminish the resistance of the animal organism to it, for it helps to explain how the disease can be more virulent in one outbreak than in another. Young calves below the age of six months seem to possess a relatively high degree of immunity, but it is by no means absolute. I dare say many of us have seen the disease affect unweaned calves. The period of life at which cattle are most susceptible is from nine months to the third year. After the third year the susceptibility diminishes, but again the degree of immunity enjoyed by older animals is not absolute.

According to Messrs Arloing, Cornevin, and Thomas, the animals bred in black-quarter districts very often have a high degree of immunity, and they suggest that these animals may have acquired this as the result of one or two mild attacks of the disease.

Virulent Matter.—As regards the virulent material, the juice of the affected muscles and the œdematous fluids contain the microbe. The same applies to the fluids from the cavities. The pulps of internal organs are virulent, and so are the bloody discharges from the nostrils and anus. It is chiefly by the latter that the pastures are reinfected. The blood contains few microbes. That it does contain a few, however, has been shown by Nocard.

As the subject of this paper is prevention, I would like here to call your attention to one measure that is entirely neglected, viz., disinfection of the spot where the animal is found dead. One cannot find out every part where the patient has been, but the places where they have lain down to die will be the most contaminated. It has been my habit for several years to advise disinfection of these spots, but I have never received any communications bearing on its efficacy. The best agents for this purpose are corrosive sublimate, 1–2000 in water; or carbolic acid, 5 per cent. aqueous solution.

Infection.—It has been experimentally demonstrated that the black-quarter virus (dried muscle juice introduced under the skin or by the alimentary tract), when a sufficient quantity is used, gives the disease to susceptible animals.

Inoculation into the muscular tissue is still more certain.

Injection into the trachea or veins excites only a rise of temperature and slight indisposition. After recovery the animal is endowed with the highest degree of immunity. Unfortunately this method cannot be applied in practice, because, should a drop of the bacterium or spore containing fluid get into the subcutaneous tissues, a fatal termination from black-quarter may be the result. Further, if while the germs of the disease are circulating in the blood a bruise be inflicted on the animal, so that some blood escapes into the tissues, the animal may die of black-quarter, just as if it had been inoculated subcutaneously from without (Arloing, Cornevin, and Thomas).

The blood, however, is a bad medium for the anærobic bacilli, and they do not flourish in it. Still, during a natural attack of the disease they seem to invade the blood-stream in small numbers, too few to be demonstrated in cover-glass preparations, though the blood can yet serve for the inoculation of culture media. As further evidence of their existence in the blood-steam, we have the occurrence of tumours away from the seat of inoculation and the presence of bacilli in the cavities. On *a priori* grounds, then, natural infection seems to operate by ingestion of spore-contaminated material, or by the spore gaining access to the subcutaneous tissues through a wound. I incline to think that the latter method is the more common, because the disease is relatively rare in coated animals like the sheep, although it is the most susceptible of all to inoculation.

Infection does not operate equally well in every part of the body.

We seldom get evidence of the virus having penetrated by those parts where the connective tissue is dense, and it has been shown experimentally that it is more difficult to obtain positive results by inoculating at parts such as the tail or the ear.

In the practice of preventive inoculation advantage has been taken of this observation. Fewer animals die from the inoculation when performed at the extremity of the tail. However, this method has its disadvantages. Accidents similar to those which followed preventive inoculation against pleuro-pneumonia are said to be fairly frequent. Moreover, it is not easy to insert a needle at this part, both on account of the movements of the animal and the density of the tissues. The other parts where the operation is practised are behind or in front of the shoulder.

Methods of Preparing the Vaccine (Arloing, Cornevin and Thomas).

The black-quarter virus in the dried state is very resistant to the action of heat. A dry temperature of 110° C. continued for six hours is necessary to completely destroy it. The same temperature when got by superheated steam destroys it in twenty minutes. Arloing, Cornevin, and Thomas say that the dry virus must be exposed to a dry temperature of 85° C. for six hours to obtain a solid diminution of virulence.

Between these ranges of temperature one can obtain virus of varying strength. To obtain their vaccine they proceed as follows. A portion of the affected muscle is bruised in a mortar with a few drops of water. The liquid containing the virus is filtered through muslin. It is then spread in a thin layer on a plate and dried at 37° C. After drying, one scrapes the plate and obtains reddish-brown scales or powder. This virus remains active for a long time. To attenuate its virulence one adds two parts of water to one of powder. The mixture is spread out on a plate in a thin layer, and exposed to a dry temperature of from 100-104° C. for seven hours. A brownish scaly material is afterwards scraped off the plate, and this is the first vaccine.

A second and stronger vaccine is obtained by exposing the virus similarly prepared to a temperature of from 90-95° C. for seven hours. The only agent that can possibly resist this temperature is the spore, but it becomes attenuated. The reddish material is powdered finely and sent out in packets. Before use, one puts the powder in a sterilised mortar (boiled fifteen minutes), and adds sterilised water (boiled) to the amount of 10 cc. The mixture is then filtered through muslin which has been previously boiled.

Arloing, Cornevin, and Thomas advise the inoculation to be performed at about 3 inches from the extremity of the tail. The dose for oxen older than eighteen months is 18 mm., and from 10-15 mm., according to size, for younger animals. The second vaccine is inoculated ten days after the first, and, of course, the operation is conducted as far as possible with antiseptic precautions.

Strebel has for some years inoculated this vaccine in the neighbourhood of the shoulder with good results, and lately it has been advised to perform the operation on the side of the chest.

Professor Kitt of Munich prepared a vaccine by exposing the virulent brown powder to a steam temperature of from 98-100° C. for about six hours. He found that sheep and oxen withstood a dose of from ·02-·5 gramme, and that afterwards they presented a higher degree of immunity. The strength of this vaccine is midway between the first and second vaccines of Arloing, Cornevin, and Thomas. Kitt's object was to endow the animals with immunity by a single inoculation, and thus reduce the trouble and expense connected with the operations. In practice, however, Kitt's method has not realised his expectations. Kitt himself has said that his own method is less surely protective than that of Arloing, Cornevin, and Thomas. Recently Kitt recommended the use of a vaccine prepared from pure cultures of the black-quarter bacillus, but the results have been by no means encouraging.

Valuable statistics of the results of preventive inoculation were presented at the Sixth International Veterinary Congress, 1895, by Prof. Cornevin of Lyons, Prof. Hess of Berne, and M. Strebel of Fribourg. I shall confine myself to the most important of them.

Cornevin records the results of 21,854 inoculations during one and-a-half years 1893-95, with a death-rate from the operation (*i.e.*, due to inoculated black-quarter)(= ·0823 per cent). He says, however, that the death-rate varied considerably in different districts. Still, in every case the operation appears to have reduced the death-rate below what it was before the introduction of preventive inoculation. He gives the mortality from black-quarter in nineteen districts before and after inoculation was introduced. That before varied from 25 per cent. to 2 per cent. Afterwards it varied from 1 per cent. to 0 per cent.

Hess records 148,569 inoculations from 1885-94. The total number of failures (*i.e.*, deaths from inoculation, or black-quarter contracted in spite of inoculation) amounts to 736 or ·5 per cent. These 736 failures are made up by numbers varying from 50 out of 13,794 in the year 1888 (= ·36 per cent.), to 84 out of 12,061 (= ·7 per cent.) in 1889.

The number of animals dead from black-quarter inoculation by the vaccine varied from 8 out of 15,137 (= ·05 per cent.) in 1885, to 36 out of 17,791 (= ·2 per cent.) in 1894. The number of deaths from black-quarter contracted naturally in spite of the inoculation varied from 31 out of 13,744 (= ·22 per cent,) in 1888, to 61 out of 12,061 (= ·51 per cent.) in 1889. One sees, then, that the number of failures is very variable, and when the bulk of them have to be recorded in one place, as sometimes happens, one's faith in the method is apt to be shaken. At least, one is diffident about advising a client to adopt it.

According to Hess the number of deaths following the first inoculation is almost two-thirds of the total number that die from inoculation black-quarter. A weaker first vaccine is then indicated, but this if prepared in the same way would probably raise the death-rate after the second inoculation, or render a third operation necessary. Lastly, Hess gives a table comparing the failures following the operations at the tail with those at the shoulder.

In 1894, 17,791 animals were inoculated, viz., 12,299 (=69 per cent.) at the tail, and 5,442 (= 31 per cent.) at the shoulder.

Inoculation at the tail gave 42 failures, viz., 17 deaths from inoculation black-quarter, and 25 from the disease naturally contracted. This gives a total of ·32 per cent., made up of ·13 per cent. from inoculation, and ·19 per cent. from natural black-quarter.

At the shoulder there were 51 failures (= ·93 per cent.), viz., 19 deaths from inoculation (= ·35 per cent.), and 32 deaths from natural black-quarter (= ·58 per cent.).

The reports by Strebel are the fullest and most interesting of all.

In Table II. he gives the results of 192,948 inoculations performed in nineteen cantons or countries from 1884-94, and he compares the death-rate from black-quarter in the vaccinated with that in 315,168 non-vaccinated animals which frequented the same pastures.

The total number of failures, from inoculation and from the disease contracted in spite of it, amounted to 922 or ·48 per cent. The highest death-rate was 30 out of 2255 animals vaccinated (= 1·33 per cent. in Uri), and the lowest was 0 out of 212 in Soleure.

Inoculation was responsible for 82 deaths (= ·042 per cent.). The highest rate was 5 out of 680 animals (= ·737 per cent.) in Haute-Autriche. The lowest rate was 0 out of 6905 in Glaris. The disease contracted in spite of inoculation was responsible for 840 of the deaths, the largest proportion being 30 out of 2255 (= 1·33 per cent.) in Uri, and the lowest 0 out of 212 in Soleure. Out of 315,168 non-vaccinated animals going on the same pastures, 5482, or 1·74 per cent., died of black-quarter. The highest rate was 13 out of 184 (= 7·06 per cent.) in Soleure, and the lowest was 89 out of 13,756 (= ·65 per cent.) in Carinthia.

In Table IV. he gives the result of 129,740 inoculations performed at the extremity of the tail, and compares the death-rate with that in 234,560 non-vaccinated animals on the same pastures.

The total number of failures was 585 (= ·46 per cent.). The highest rate was 1·33 per cent. in Uri, and the lowest 0 per cent. in Soleure.

These cases are also included in Table II. From inoculation there were 35 deaths (= ·027 per cent). The highest rate was 6 out of 3951 (= ·152 per cent.) in Vaud, and the lowest was 0 out of 6909 in Glaris. From black-quarter naturally contracted there 550 deaths (= ·42 per cent.).

The highest rate was 30 out of 2255 (= 1·33 per cent.) in Uri, and the lowest was 0 out of 212 in Soleure. Of the non-vaccinated 4136, or 1·76 per cent., died of the disease. The highest rate was 13 out of 184, or 7 per cent., in Soleure; the lowest was 89 out of 13,756, or ·65 per cent., in Carinthia.

In Table VI. the results of 62,203 inoculations performed at the shoulder are given, and the death-rate is compared with that in 82,334 non-vaccinated animals on the same pastures. The number of failures amounted to 327, or ·52 per cent.; the highest rate was 106 out of 8865, or 1·19 per cent., in Styria; and the lowest was 9 out of 6732, or ·13 per cent., in Salzbourg. The inoculation accounted for 45 deaths (= ·072 per cent.); the highest rate was 5 out of 680, or ·735 per cent. in Haute-Autriche, and the lowest 0 out of 5758 in Hungary. The disease naturally contracted killed 282, or ·45 per cent., of the vaccinated; the highest rate was 91 out of 8865, or 1·02 per cent., in Styria; the lowest 6 out of 6732, or ·09 per cent., in Salzbourg. Of the non-vaccinated 1350, or 1·64 per cent., died of black-quarter; the highest rate was 172 out of 6254, or 2·76 per cent., in Haute-Autriche; and the lowest 169 out of 17,854, or ·95 per cent., in Styria.

In Table VII. are given the results of the single inoculation method performed at the shoulder in 53,656 cases.

The number of failures was 258, or ·48 per cent. Inoculation killed 67, or ·125 per cent.; the highest rate was 12 out of 1429, or ·840 per cent., in Basse-Autriche, and the lowest 0 out of 962, in Fribourg. In spite of the vaccination 191, or ·350 per cent. of the animals, died of the disease. The highest rate was 91 out of 8865, or 1·03 per cent., in Styria; the lowest was 0 out of 12,300 in Algeria.

In Table IX. we find the results of 34,756 inoculations performed at the shoulder with the dry vaccine of Kitt (*i.e.* a single inoculation with a virus midway in strength between the first and second vaccines of Arloing, Cornevin, and Thomas). The death-rate is compared with that in 65,518 non-vaccinated animals on the same pastures. The total loss from black-quarter in the vaccinated amounted to 203, or ·58 per cent.; the highest rate being 5 out of 392, or 1·27 per cent., in Haute-Autriche; the lowest 9 out of 6732, or ·13 per cent., in Salzbourg.

The operation was responsible for 42 deaths, or ·121 per cent. The highest rate was 1·275 per cent. in Haute-Autriche; the lowest ·044 per cent. in Salzburg.

Naturally contracted black-quarter accounted for 161 deaths, or ·46 per cent. The highest rate was 1·02 per cent. in Styria; the lowest 0 out of 392 in Haute-Autriche. Of the non-vaccinated 992, or 1·51 per cent., died of the disease. The highest rate was 1·91 per cent. in Haute-Autriche; the lowest ·95 per cent. in Styria.

In Table X. we find the results of 5644 inoculations performed with the pure culture vaccine of Kitt (the virus is injected once at the shoulder).

The death-rate is compared with that in 13,183 non-vaccinated animals on the same pastures.

In the vaccinated the total loss was 89, or 1·58 per cent. The inoculation killed 8, or 1·41 per cent.; the disease naturally contracted killed 81, or 1·43 per cent.

In the non-vaccinated, 191, or 1·45 per cent., died of black-quarter.

Lastly, Strebel gives a Table (XI.) in which are compared the results obtained from the different methods.

[TABLE.

Method.	*No. of Animals Vaccinated.*	*Died.*					
		From Inoculation.	*Per Cent.*	*From naturally Contracted Black-Quarter.*	*Per Cent.*	*Total.*	*Per Cent.*
Double inoculation at tail	325,893	188	0·056	1245	0·38	1430	0·44
Total inoculations in region of shoulder	91,066	76	0·084	365	0·40	441	0·48
Double inoculation in region of shoulder	37,410	8	0·022	157	0·42	165	0·44
Inoculation (single) behind shoulder with the dry vaccine of Kitt	39,084	61	0·156	187	0·48	248	0·63
Single inoculation with the pure culture vaccine of Kitt	5,643	8	0·141	81	1·43	89	1·58

Some apparent relation can be traced between the severity of the inoculation and the number of animals which contracted the disease naturally afterwards. As a rule, where the death-rate from inoculation is high, the number of failures from insufficient protection is relatively small.

These percentages are very valuable, but I do not think that the laws of proportion can be given their full arithmetical significance in a case of this kind, because there are several factors, such as variation in virulence and susceptibility, about which we have too little information. We know that in certain parts the operations have been followed by many fatal results, that on this account the practice of preventive inoculation against black-quarter was abandoned in England, and that the degree of immunity derived from it has been in many cases insufficient to ward off a spontaneous attack of the disease. The latter part of the resolution voted by the Sixth International Veterinary Congress appears to me to be an excellent criticism on the applicability of the method ; it is as follows :

"The Veterinary Congress regards preventive inoculation against black-quarter, discovered by Messrs Arloing, Cornevin, and Thomas, as a precious means of combating the disease, and practicable in the countries where compensation provides for the losses following inoculation."

Setoning as a Preventive of Black-Quarter.

One of the oldest measures directed against the disease in this country is setoning. The operation is known to all ; I need say nothing about that part of it. I have been unable to discover who was the originator of the idea,[1] nor have I met with any statistical report on its efficacy. However, it is widely practised at the present day in black-quarter districts. Many veterinarians of long experience put great faith in it as a preventive, others practise it on the grounds that they must do something, and setoning is the only means they have at hand. I think I may say that the majority of practitioners put some faith in it, at least such an opinion I have come to after many inquiries.

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[1] The seton method is mentioned by John Lawrence in "A General Treatise on Cattle," 1809. Francis Clater "Every Man His Own Cattle Doctor," 1825. Steel "On the Ox," 1881.

They say that experience has taught them that the disease frequently stops after a herd has been setoned. They have no explanation to offer regarding its action; they simply fall back on their experience.

This method of reasoning may well be called in question, especially when dealing with a disease like the one under discussion, for everyone knows that a single animal in a herd may die of black-quarter and its companions remain healthy whether they are setoned or not. Evidently the disease is not very contagious in the ordinary sense of the word. It does not pass from animal to animal as pleuro-pneumonia seems to do; it is to be classed rather with such bacterial diseases as tetanus. There are three tests which might have been applied to the method. Firstly, that of comparing the death-rate in a black-quarter district for a number of years before setoning was introduced, with that during several years after its adoption. This, however, would have given misleading results in many districts, because the disease has almost entirely disappeared from certain farms on account of improved treatment of the land. Secondly, a number of setoned animals might have been pastured with the unsetoned on black-quarter farms, and the death-rate from black-quarter compared in the two sets of animals. To make such a test of any value a great number of animals would have to be kept under observation over several years, and there would have to be a decided difference in the mortality of the two sets of animals before an opinion could be formed one way or the other. The third test is one which according to our present notions should be the initial one. It is to inoculate with equal doses of the same virulent material one lot of susceptible animals that have been setoned, and a second lot not setoned but otherwise kept under the same conditions as the first, in order to see whether the setoned lot withstand the inoculation entirely or for a longer period than the unsetoned. A combination of the second and third tests would be the most satisfactory of all. My investigations have dealt entirely with the experimental test.

It appeared to me that one could not disregard entirely the evidence of experience, such as it is. I did not think that the mere setoning could possess of itself any virtues, but with others I thought it possible that the products of the microbes of the suppuration which follows the operation might have the property of endowing the animal organism with some degree of immunity. This idea is not purely imaginary. Roux has shown that animals rendered immune against black-quarter itself are also immunised against septicæmia. The Midland Veterinary Association in 1891 put the setoning method to an experimental test, while they were conducting other experiments on the efficacy of protective inoculation with the Lyon's and the Kitt vaccins. When I began my experiments I did not know that the efficacy of the setoning method had been tested in any way, but I purposely selected my animals from a very susceptible species, viz., the sheep, and was thus spared the disappointment of seeing the control animals survive the others except in one case. In the experiments undertaken by the Midland Veterinary Association the setoning method was tested on calves, animals whose susceptibility is very variable. Indeed, it cannot be denied that the majority of calves possess a high degree of immunity from black-quarter. The result of the above-mentioned experiments was to leave the question practically in *statu quo*. My first experiments were undertaken to see if the products of pyogenic microbes had any protective action. I obtained two yearling sheep (ewes). One was kept as a control, the other received hypodermically on the 24th November 1896, at 1.30, 6 cc. of an eleven-days'-old broth culture of the staphylococcus pyogenes aureus from which the microbes had been removed by passing it through a porcelain filter. The normal temperature of this ewe was 104° F. There was no rise of temperature after injection. It is needless to say that the surgical operations in all cases were performed with strict antiseptic precautions.

I thought it might be of interest to observe if the dissolved products of the staphylococcus aureus had any effect on the number of white cells in the blood, for everyone knows the important part assigned to the phagocyte in the production of immunity. The colourless corpuscles in the blood of this animal numbered 18,400 per cub. mm. before the experiment.

26th November, 11 A.M. 10 cc. of same toxin (eleven days' culture) were injected hypodermically. 12 P.M., temperature 104·6°. White cells 16,800 per cub. m.

27th November, 1.30 P.M. 12 cc. of same toxin injected. White cells 16,800 per cub. m.

28th November. Temperature, 104° F.

29th November, 11 A.M. 16 cc. of same toxin injected directly into jugular vein.

12 P.M. Temperature, 105° F. White cells 17,600 per cub. m.

1st December, 12 P.M. 20 cc. of same toxin into jugular vein.

12.30 P.M. White cells 16,000 per cub. m. (vein).

6th December. Temperature, 104° F. 30 cc. of filtered broth culture of staphylococcus pyogenes aureus, after fourteen days' incubation, were injected into jugular vein, and 4 cc. hypodermically. (This culture was made from the pus of an experimentally produced abscess.) White cells twenty minutes after the injection numbered 9600.

I do not wish to lay too much stress on these observations on the white cells, since I have not made a sufficient number of observations. So far, however, they seem to show that injection of large quantities (30 cc.) of pus toxin into the veins, is followed by a temporary diminution in the number of white cells of the blood. From the 24th November to the 6th December this ewe received 98 cc. of pus toxin, 32 cc. under the skin, and 66 cc into the veins.

10th December. 5 cc. of sterile bouillon were mixed with the subdermic fluid of a guinea-pig dead of black-quarter, and 1 cc. of this mixture was injected into the shoulder of the ewe.

11th December, 10.30. Ewe very ill, cannot stand, temperature 105° F., comatose. The animal died of black-quarter some time in the evening of the 11th.

On the 10th December a control yearling ewe, which had been kept all the time under the same conditions as the other, except that it received no pus toxin, was inoculated with 1 cc. of the same mixture of bouillon and black-quarter virus. The temperature of this animal rose from 104° F. at the time of the injection to 105° F. on the next day. There was no perceptible tumour at the seat of inoculation, and the animal remained alive and well.

It may be objected that the control ewe had a greater natural resistance than the other. If it had, the experiment at least shows that this resistance was greater than that conferred on its fellow by the pus toxin, which was nil.

I am inclined to believe, however, that the pus toxin lessened the natural resistance of the other animal. I may say, that I tried afterwards to overcome the now reinforced immunity of the control by injecting pus toxin, but failed. I have also performed three similar experiments on guinea-pigs, using the staphylococcus pyogenes aureus itself as well as its products obtained by filtration. In no case have the pus microbes or their products prolonged the life of the animals injected with black-quarter virus.

I thought, however, that it would be more satisfactory if experiments were performed on animals that had been actually setoned, since their condition would then correspond to that of animals presumably immunised by setoning, for it appeared quite possible that the animals might derive some degree of immunity from microbes other than the staphylococcus pyogenes aureus.

Accordingly, I obtained three healthy yearling ewes. One of them (A) was setoned in the neck, according to the usual method, without any antiseptic precautions. Another (B) was setoned according to the rules of antiseptic surgery, and the wound was kept aseptic by covering with a pad of corrosive sublimate wool, which was soaked every day with creoline wash. At no time during the experiment was there any sign of suppuration in this animal. The third ewe (C) was kept as a control. The animals were lodged in a roomy loose-box, and fed on oats, maize, bran, hay, and grass.

The setons were allowed to act for a month. The white cells of the blood were counted before and at different stages of the experiment, but they underwent no appreciable change.

On the 6th July each of the three animals received into the left thigh 1·7 cc. of an emulsion made by triturating in a sterilised mortar ·5 gr. of dried black-quarter muscle with 8 cc. of sterile water, and adding one drop of acetic acid after straining through muslin. The muscle was about one year old. The details of these experiments are as follows:—

Ewe (A) (suppuration.) 6th July, 12.30 P.M. Temperature, 103°. Injected 1·7 cc. of above emulsion into muscles of left thigh.

7th July, 11.0 A.M. Temperature, 105·6°. Leg swollen and livid at and below seat of inoculation, but not much emphysema. Animal very dull and partially comatose, could not stand, apparently dying.

Died of black-quarter at 11.30 A.M.

Ewe (B) (aseptic seton.) The object of this experiment was, of course, to see whether microbes were responsible or not for any degree of immunity that might be produced.

6th July, 12.30 P.M. Temperature, 104° F. Injected 1·7 cc. of same virus (as above) into muscles of left thigh.

7th July, 11.0 A.M. Temperature, 106°. Leg swollen and livid at and below seat of inoculation. Very lame but walking about, slight emphysema of thigh, breathing hurried.

9.0 P.M. Dull but able to walk about, does not look like dying.

11.30 P.M. Able to walk, no signs of coma.

8th July, 10.30 A.M. Temperature, 106°. Still no sign of coma, able to walk.

Died of black-quarter at 1.30 P.M.

Ewe (C) (control). 6th July, 12.30. Temperature, 104·5° F. Received 1·7 cc of same emulsion (virus) into muscles of left thigh.

7th July, 11.0 A.M. Temperature, 105° F. Leg swollen and livid at and below seat of inoculation. Lame but not so bad as others, still runs off when approached, slight emphysema at seat of inoculation.

9.0 P.M. Very ill, partially comatose. Died of black-quarter at 11.0 P.M.

It will be seen that (A) (septic seton) died in the short space of twenty-four hours, and predeceased (C) (control) by twelve hours, and (B) (aseptic seton) by twenty-four hours, while (C) died about eleven hours before (B).

The relatively early death of (A) is in accordance with the result of the experiments performed with pus toxin. The results are sufficient to convince me that setoning does not endow animals with any degree of immunity from black-quarter; in fact, there is some evidence to show that, as performed in practice, it has the opposite effect. I feel sure that if further experiments be undertaken on the same lines, the evidence against the efficacy of setoning will only become the stronger.[1] Were it possible to determine exactly what is the fatal dose of the black-quarter virus, one might experiment with greater exactitude. On account of the varying degrees of individual susceptibility and for other reasons this cannot be done. The virus which I employed, however, cannot have been of excessive strength, because at the same time I inoculated ·5 cc. of the same virus into the thighs of two guinea-pigs, one protected by filtered muscle juice, the other not, without a fatal termination in either case.

I have also performed some experiments to establish the degree of immunity conferred on a susceptible animal by the filtered muscle juices from animals dead of black-quarter. When I began the latter experiments I was not aware that Roux in 1888 (*Annales de l'Institut Pasteur*) had already shown that immunity could be thus conferred. Fortunately I came across this paper before proceeding very far. Roux showed that if the affected muscles of guinea-pigs dead of black-quarter be rubbed up with half their weight of water, the liquid obtained after pressing and filtering through porcelain could confer immunity on other guinea-pigs, if injected in large doses. According to the same authority cultures of the bacillus sterilised by heating to 115° C. had the same effect, but the immunity derived from the juices was superior. This subject was again taken up by Duenchmann (*Annales de l'Institute Pasteur*) in 1894. Experiment-ing with guinea-pigs, he was able to confirm Roux's finding with regard to the filtered juices, but could not do so regarding the filtered cultures. His results with the latter substances led him to conclude that the toxines from cultures were the reverse of protective. This is quite in accordance with the view that the products of the bacilli, acting like lactic acid, etc., help to overcome the resistance of an animal to black-quarter. I was proceeding in a similar, though slightly different manner. By means of a meat press I obtained the juices from the affected muscles of sheep. These juices were passed through a Chamberland filter, and then tried on guinea-pigs and sheep. The results of my experiments have been to confirm what Roux had already found in the guinea-pig, viz., that the muscle juices of animals dead from black-quarter are protective for other animals after filtration.

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[1] Since the reading of this paper I have received a very valuable observation from my friend Mr F. of B., who has taken much interest in this investigation. At the request of a client, whose losses from black-quarter are annually very high, my friend setoned fifteen yearling beasts. For some reason a sixteenth beast was not setoned. The sixteen animals were all pastured on the same meadows. All the setoned animals died of black-quarter, and were survived by the one that had not been setoned.

My object in undertaking the latter experiments was to see if it were possible to obtain a substance practically harmless to animals, but capable of enabling them to withstand such a dose of strong virus as would endow them with a higher degree of immunity than that acquired from the methods of preventive inoculation now practised. I have already pointed out that failures occur either because the vaccine is too strong or too weak. My reasons for using the muscle juice were, that it appeared to me to be the best way of getting an active toxin, and that one would get the benefit of any antitoxin substances that might be formed in the animal organism. If it were possible to get in the muscle juice such a protective substance as I have spoken of, one would have no hesitation in availing oneself of preventive inoculation even in districts where the death-rate from black-quarter is relatively very small. The amount of experimental evidence which I possess on this point is not sufficient as yet to justify a communication. So far, however, the results have been encouraging, and I hope at a future time to have the pleasure of communicating the outcome of some further experiments.

A FURTHER NOTE UPON MEDIAN NEURECTOMY.

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IN the *Journal of Comparative Pathology and Therapeutics* for September 1896, there was published an article upon "Median Neurectomy" in which was given a description of the method of operating and a summary of thirty-six cases in which the operation had been performed.

In the present article I propose to trace up as far as possible these cases, and to give an account of an additional fifty-one cases, making a total of eighty-seven individual animals (consecutive cases). The majority of them were patients in the Free Clinique of the College, and were operated upon by students of classes C or D, the remainder being operated upon after consultation, and in conjunction with practioners in private practice.

It must be acknowledged that if the animals which have been operated upon have stood the test of time and been made useful for a prolonged period of work, "Median Neurectomy" may justly be considered amongst the operations which we should utilise for the relief of certain forms of lameness in the ordinary routine of daily practice. Particularly is this the case when the simplicity of the operation is considered.

In many of the cases operated upon all other known methods of treatment had previously been tried without success, and the only alternative course was slaughter.

Referring back to the cases recorded in the Journal for September 1896 the termination of Cases 1, 5, 6, 7, 10, 17, 23, 25, and 27 have been already mentioned.

CASE 2.—Cab mare operated upon 16th October 1895, worked well and regularly until October 1896, when she met with an accident and was killed.

CASE 3.—An aged cab mare, operated upon 29th October 1895, worked regularly in the owner's possession until August 1896, when she was sold. The owner saw the animal at work for some time afterwards, but since then I have not been able to trace her.

CASE 4.—A high stepping pony, six years old when operated upon on the 16th of November 1895, is still working regularly and well, and has not been lame since. There is no alteration in the action, the pony stepping as well as ever.

CASE 8.—A carriage mare, about nine years old when operated upon on the 3rd of February 1896, has not suffered from lameness on that leg since, and is at present working regularly in a brougham.

CASE 9.—An aged cab mare, operated upon 5th February 1896, is still working regularly and has not been lame on that leg since.

CASE 11.—A cab gelding, nine or ten years old when operated upon on the 29th of February 1896, is still working regularly every day, and has not been lame on that leg since.

CASE 12.—A cart mare, six years old when operated upon on the 16th of March 1896, is still working regularly and well. A large splint which was present under the knee is perceptibly diminished in size.

CASE 13.—An aged van gelding, operated upon on the 18th of March 1896, is still working regularly and has not been lame on that leg since.

CASE 14.—An aged van gelding, operated upon on the 2nd of April 1896, in both fore legs, has been working regularly ever since.

CASE 15.—A very old pony, operated upon on the 2nd of April 1896, has been working regularly ever since.

CASE 16.—An aged cab mare, operated upon on both fore legs, 20th April 1896, has worked regularly without lameness ever since.

CASE 18.—A cab mare, aged, operated upon 25th April 1896. When publishing the account last year I had been unable to trace the case, but since then I have seen the owner and find that the animal has been working regularly ever since.

CASE 19.—An aged cab mare, operated upon on the 27th of April 1896, is still working regularly and has shown no return of the lameness.

CASE 20.—An aged cart gelding, operated upon on the 29th of April 1896, is still working regularly and has shown no return of the lameness.

CASE 21.—An aged cab gelding, operated upon on the 29th of April 1896, has been working regularly in a hansom ever since.

CASE 22.—An aged cab gelding, operated upon on both fore legs, 29th May 1896, was working regularly and well up to February 1897. On the 19th of that month the animal was brought to the Clinique suffering from a wound of the off-fore coronet which appeared to be the result of a bruise or tread; on the 24th this was going on well and had almost healed. The animal went to work again after this, but was eventually destroyed in June owing to a suppurating corn which terminated in quittor.

CASE 24.—An aged cab gelding, operated upon on the 15th of June 1896, worked regularly and well until December, when the animal fell lame again on the same leg, appearing in great pain and continually resting the leg. An examination of the seat of operation revealed excessive tenderness on pressure, and a distinct lump as large as a hazel nut apparently adherent to the fascia at the back of the radius. A neuroma was diagnosed and removed, after which the horse walked without lameness, and trotted almost sound at once. The wound progressed favourably, and everything promised well for a good recovery. Unfortunately, the animal bruised its elbow whilst standing in the stable, and as this suppurated and looked like being a prolonged case, the owner had the horse destroyed without my knowledge.

CASE 26.—A van mare, nine or ten years old when operated upon on the 22nd of June 1896, has been working regularly ever since.

CASE 28.—An aged cab mare, upon which median neurectomy was performed 17th July 1896, and double plantar neurectomy 7th August, worked regularly and well until 16th October, when the animal was condemned for farcy and destroyed.

CASE 29.—An aged cab gelding, operated upon on the 17th July 1896, is still working regularly.

CASE 30.—An aged cab gelding, operated upon in both fore legs on the 29th July 1896, with the exception of about three weeks' rest worked regularly until June 1897, when a fall in the street caused a fractured limb, for which the animal was destroyed. The three weeks' rest occurred in November and December and were due to lameness caused by very severe thrush involving the sensible structures of one fore foot; treatment was adopted and the animal returned to work as usual.

CASE 31.—A cart gelding, operated upon (when seven years old) on the 29th of July 1896, showed decided improvement in the lameness when last seen (21st October), but was still too lame for work. External plantar neurectomy for the relief of a ringbone, which had developed since the date of the median operation, was advised, but the owner would not give consent. The animal was turned out to grass for a while, after which the lameness completely passed off, and it is now working regularly. Once or twice it has shown lameness since on the same leg.

CASE 32.—A cab mare, operated upon (when eight or nine years old) on the 10th of August 1896, showed improvement after the operation. External plantar neurectomy was advised for the ringbone present, but the owner did not give consent, and since then I have not been able to trace the patient.

CASE 33.—An aged trap gelding, which had median neurectomy performed 13th August 1896, and external plantar neurectomy on the 28th, improved sufficiently to go to walking or slow trotting work in September. The animal improved more at work, and has worked regularly ever since.

CASE 34.—An aged cab gelding, operated upon on the 21st of August 1896, was sent to work early in September, but I have been unable to trace it further.

CASE 35.—A pony gelding, six years old when operated upon on 21st August 1896, did not show any immediate improvement after the operation, but early in September was sufficiently improved to be able to do walking work. Since then I have been unable to trace it.

CASE 36.—An aged cab mare, operated upon on the 26th of August 1896, is at present working in an omnibus. With the exception of a few weeks' rest, due to a wound (on the same leg) caused by the shaft of a cart, the animal has been working regularly ever since.

The above gives the history, as far as possible up to date, of the cases that were published last year in the September number of the Journal. The following are those which have been operated upon since:—

CASE 37.—Cob, aged, very lame. Suffering from a large exostosis on the inside of the carpus. Before the operation was performed the owner was told that the lameness was probably partly mechanical owing to the situation of the bony growth.

10th September 1896. Median neurectomy under chloroform. There was a decided improvement after the operation, and when the animal was seen a month later this improvement was maintained. Unfortunately I have been unable to trace the case further, and cannot say whether the animal ever became fit for regular work.

CASE 38.—Cart mare, rising six years old, suffering on the off foreleg from chronic tendinitis, a chain of splints inside the cannon bone, and a large exostosis on the knee, involving both the front and side. The tendons were thickened and contracted. This was the case operated upon before the members of the North of England Veterinary Medical Association. The mare had been lame for about ten months, and was fired and blistered without improvement three months ago. She could not do work without being lame for some days afterwards. The lameness was diagnosed as being partly due to the splints (as they were tender on pressure), and partly mechanical owing to the exostoses on the knee joint; the latter interfered with the joint so much that it could not be properly flexed.

20th November 1896. Median neurectomy under chloroform. Immediately after the operation there was a decided improvement; the wound healed well, and the animal was used for walking work until January 1897, after which she gradually became lame again, and at present, Mr Elphick informs me, seems in much about the same condition as before the operation. The enlarged and stiffened condition of the knee joint is undoubtedly the chief cause of this, the lameness being mechanical.

CASE 39.—Cab mare, aged, had been very lame for about a month. The animal had a chain of splints under the tendons, chronic tendinitis, and ringbones. Lameness was diagnosed as being due to the chronic tendinitis and splints.

18th November 1896. Median neurectomy under chloroform. Lameness only very slight after operation. The wound progressed favourably, and the animal went to work early in December. There has been no return of the lameness, and the patient has worked regularly ever since.

CASE 40.—Light van mare, aged, had only been in owner's possession about three weeks, and had been very lame all the time. There was a very large splint, a sidebone and a ringbone on the inside of the leg, and a small ringbone on the outside. The splint was very sore on pressure, and lameness was diagnosed as being due to the irritation produced by this.

25th November 1896. Median neurectomy under chloroform. No lameness could be perceived after the animal had trotted up and down a few times. This improvement was maintained, and the animal worked well for about a month, when it met with an accident in the street and was destroyed.

CASE 41.—A light van gelding, aged, had been in the owner's possession about ten months, and been very lame a month. There was a large ringbone, a large splint, and chronic tendinitis. The lameness was diagnosed as due to the latter, and to the irritation produced by the splint.

26th November 1896. Median neurectomy under chloroform. There was a perceptible improvement after the operation, but the animal never became sufficiently sound for the work required, and was sold at the end of two months; perhaps the ringbone may have been partly the cause.

CASE 42.—Cab mare, seven or eight years old, had been lame some weeks. Had ringbone and a large splint about the size of a cob-nut under the tendon of the near fore leg. The splint was very tender on pressure, and had been severely punctured with the firing iron some time before. Lameness was diagnosed as being due to the presence of this splint.

30th November 1896. Median neurectomy under chloroform. During the progress of the operation the student accidentally cut a collateral branch of the radial artery; this was ligatured and the operation completed. It was too dark to see whether the lameness had disappeared or not. The animal was seen again on the 9th and 15th of December, and was decidedly better when walking, but showed lameness when trotting. The wound, however, was granulating freely, and had to be cauterised. Eventually she recovered and was worked by the owner for two months, but as she frequently caught the toe on the ground and stumbled he sold her for £6. Since then I have been unable to trace her.

CASE 43.—Pony, nine or ten years old, suffering from chronic tendinitis, contracted tendon, overshot fetlock, and ringbone. The animal was very lame both when trotting and walking, and had been so for three months.

30th November 1896. Median neurectomy was performed as a last resource. It was too dark to note progress immediately after the operation.

30th December. The animal was sent to walking work, but was very lame when trotting.

20th January 1897. In order to make a further attempt to preserve the animal and make it fit for trotting work, external plantar neurectomy was performed. As, however, this did not have the desired effect the owner sold the animal, and since then I have been unable to trace it.

CASE 44.—A cab mare, aged, had had double plantar neurectomy performed on both fore legs for ringbones some nine months before. She had worked well until about a week ago, and was now suffering from very painful tumours on the ends of the divided nerves. As the legs were very puffy it was decided to try the effect of median neurectomy instead of dissecting out the tumours.

7th December 1896. Median neurectomy both fore legs. There was a decided improvement after the operation.

1st January 1897. No lameness. The wounds healed, but the owner had noticed a swelling in the off fore heel during the last week. On the 12th it was plain that gelatinous degeneration was taking place, and on the 14th the animal was destroyed.

CASE 45.—Cab horse, was suffering from navicular disease, and had had double plantar neurectomy performed on the 26th of October. At the present time the animal was suffering from lameness, especially noticeable at the commencement of a journey. There was also a large splint under the tendons, and the lameness was diagnosed as being due to this.

21st December 1896. Median neurectomy under chloroform. Immediately after the operation there was a perceptible improvement, and on the 30th this was more marked. The animal was sent to work on the 11th of January, and has been working regularly since without lameness.

CASE 46.—A cab gelding, aged, had been lame off and on for eighteen months. The animal was suffering from a chain of large splints and ringbones on the near fore leg. Actual cautery was applied without any improvement five weeks before.

13th January 1897. Median neurectomy under chloroform. After the operation there was no lameness whatever, and since then the animal has worked regularly without any return of the intermittent symptoms.

CASE 47.—An aged pony, excessively lame on the near fore leg, due to the presence of a large splint under the tendons, and slight lameness on the near fore due to a chain of splints. The owner reported that the animal had been lame off and on for about six months.

13th January 1897. Median neurectomy on the off fore under chloroform.

14th January. Median neurectomy on the near fore. After the two operations there was no lameness whatever.

28th January. The wound was going on well, but the animal was exceedingly lame again.

6th February. As on 28th January, but the wound had now healed. Diagnosis of sprain of the pectoralis transversus muscle was made, and the parts thoroughly blistered. In about a fortnight afterwards recovery took place, and the animal worked regularly ever since until three weeks ago, when it fell lame from spavin on the near hind leg. This has been fired, and the animal is at present (September) out at grass.

CASE 48.—Aged pony, had been very lame on the off fore leg for two months, suffering from the effects of an exostosis as large as an average-sized orange on the inside and front of the knee.

18th January 1897. Median neurectomy under chloroform. Noticed peculiar rigors of the limb when the animal got up. Immediately after the operation there was no lameness when walking, and after trotting for about 100 yards no lameness could be perceived at that pace. The animal was sent to work about the 10th of February, and since then has worked regularly without showing any lameness.

CASE 49.—An aged cab gelding, had been very lame off and on for six months on the off fore leg. Was suffering from a chain of splints and ringbones, both of which had been fired about three months before with temporary relief, but a full day's work always caused a return of the lameness.

18th January 1897. Median neurectomy under chloroform. When the animal stood up peculiar rigors of the limb were noticeable, as in Case 48. When walked and trotted there was no lameness. The horse was sent to work about the 11th of February, but since then I have been unable to trace its progress.

CASE 50.—Cab mare, seven or eight years old, had been very lame for some months, was suffering from a chain of large splints and ringbones.

19th January 1897. Median neurectomy under chloroform. Immediately after the operation the lameness was scarcely perceptible. The animal was sent to work on the 14th of February, and has been working regularly ever since.

CASE 51.—Cab mare, aged, very lame from a large exostosis on the inside of the fetlock joint; also had a splint and ringbones. A very bad case.

26th January 1897. Median neurectomy under chloroform as a last resource. There was a slight improvement after the operation, but lameness was undoubtedly largely mechanical. I have been unable to trace this case further, so cannot definitely state whether the animal recovered sufficiently for walking work or was killed.

CASE 52.—A pony, seven years old when operated upon (3rd February 1897). The animal had been lame on and off for five months, and was suffering from a chain of small splints under the knee. These had been treated by several applications of blistering ointment without success. After the operation there was no lameness when walking, and when trotting it was only perceptible to an expert. The patient was sent to work at the end of February, and worked regularly for some time. Since then it has been sold and lost sight of.

CASE 53.—Aged cab gelding, suffering from intense lameness due to a large splint under the tendons of the near fore leg; the animal had been in the present owner's possession for four months, and had been lame off and on the whole time; ringbones were also present.

3rd February 1897. Median neurectomy under chloroform. After the operation there was no lameness. When put to trotting work early in March the animal still showed lameness, and a smart blister was applied over the seat of operation. External plantar neurectomy was also advised, but the owner would not consent. The animal recovered sufficiently for walking work, and was sold; since then (June) it was kicked by another horse and sustained a fractured leg, for which it was destroyed.

CASE 54.—Cab mare, aged, has been lame four months, suffering from an exostosis inside the fetlock joint and ringbones. The actual cautery had been applied to the exostosis at the commencement of the lameness, but without benefit.

5th February 1897. Median neurectomy under chloroform.

15th February. Still very lame; probably largely mechanical. Advised external plantar neurectomy, but the owner would not consent until the wound had healed completely.

19th February. Wound had healed, but was now granulating a little too freely. The lameness to-day was scarcely perceptible. The mare made a good recovery, and has been working regularly ever since.

CASE 55.—Cab mare, aged, had been in owner's possession two years, and had been lame on the near fore leg off and on for eighteen months. There was a large exostosis under the knee, an atrophied frog, and a concave sole. The lameness was diagnosed as being due to navicular disease, and the ordinary double plantar neurectomy was performed on the 8th of February 1897. This produced no improvement, so, thinking that the exostosis under the knee was probably a source of pain, the animal was again cast, and median neurectomy performed under chloroform. When trotted immediately after the operation there was no lameness. Owing to the upper wound not being kept clean a pocket of pus formed under the arm; this was lanced, and on 9th March, although the leg was still swollen and the wound discharging, there was but very slight lameness. The mare, however, never got sound enough for cab work, and was destroyed.

CASE 56.—Cab mare, been very lame about two months, suffering from a large splint and side bone on the inside of the leg, and a thickening on the inside of the fetlock joint.

10th February 1897. Median neurectomy under chloroform. After the operation the lameness was scarcely perceptible. The wound healed well, but about three weeks afterwards the mare bruised her knee very severely in the stable. This suppurated and burst, leaving a very nasty ulcerating and excessively granulating wound on the front of the joint. This was successfully treated, and the animal sent to work about 12th April. Since then there has been no return of the lameness, and the animal has worked regularly.

CASE 57.—Cab mare, aged, has been very lame on both fore legs for about a month; has large splints and ringbones on both fore legs; has been fired on the ringbones; the splints show great tenderness on pressure.

18th February 1897. Median neurectomy on both fore legs, under chloroform. Immediately after the operation there was no lameness. The wounds healed well, and the animal was sent to work about 15th March. Since then there has been no lameness, and the animal has worked regularly.

CASE 58.—Cab gelding, six years old when operated upon (18th February 1897); has been lame for three months, and has a large splint, a ringbone, and a seedy toe. Median neurectomy was performed, but without any immediate beneficial result. On the 9th of March external plantar neurectomy was performed, but the animal was still, on the 27th of March, too lame for work. Since then, I have been unable to trace the case.

CASE 59.—Cab mare, aged, had only been in the owner's possession a fortnight, and had been lame all the time; has a large exostosis (tender to pressure) under the knee, chronic tendinitis (probably largely due to the exostosis), and ringbones. The latter had been fired at some time or other.

19th February 1897. Median neurectomy under chloroform. There was no immediate improvement, but the animal eventually recovered about the 12th of March, and is at present doing regular work without showing any lameness.

CASE 60.—Cab gelding, aged, very lame on both fore legs. The lameness was diagnosed as being due to exostoses on the inside of each fetlock joint; ringbones were also present on each leg and a large splint on the off.

22nd February 1897. Median neurectomy was performed on both fore legs. After the operation there was a decided improvement, but the animal was still too lame to work, owing to the off fore leg; there was no perceptible lameness on the near fore.

15th March. External plantar neurectomy was performed on the off fore leg. As there was still no improvement, as a last resource the internal plantar nerve was divided, and subcutaneous periosteotomy performed on the exostosis under the knee. There was no improvement a fortnight later, and the animal was destroyed.

CASE 61.—Cab mare, aged, had been very lame for six weeks; was suffering from lameness due to an exostosis on the inside of the fetlock joint. This had been fired without benefit.

9th March 1897. Median neurectomy was performed. The animal never recovered sufficiently to do trotting work, and after being kept by the owner for about two months was sold to someone who could use it for walking work. Before the operation the mare was very lame even when walking slowly. Since then I have been unable to trace the case.

CASE 62.—Cab mare, aged, had been very lame for two months. There was present an exostosis on the inside of the fetlock and a ringbone. Lameness was diagnosed at the time as being due to the exostosis, which had already been fired and blistered without benefit.

11th March 1897. Median neurectomy under chloroform. There was no lameness after the operation. The wound healed without much trouble and the animal is still working; the owner states, however, that the mare occasionally falls lame after a severe trot, but that when worked quietly there is no return of the lameness.

CASE 63.—Cab gelding, aged, has been very lame for a month. Lameness was diagnosed as being due to chronically sprained tendons; the latter had been fired at some time or other.

12th March 1897. Median neurectomy was performed, and after the operation there was no lameness.

19th March. Animal going well; no lameness. Since that date I have been unable to trace the case.

CASE 64.—Cab mare, aged, suffering from lameness due to chronically sprained tendons and an exostosis on the inside of the fetlock joint. These parts were severely fired and blistered about two months ago without benefit, the animal having only been able to do three days' work since, as she falls lame after being at work for a few hours.

12th March 1897. Median neurectomy was performed. Immediately after the operation lameness was hardly perceptible, and it passed off completely after trotting a short distance.

The mare was sent to work early in April and has been in regular work ever since without showing any more lameness.

CASE 65.—a hunter mare, aged, suffering from splints, exostosis on the inside of the fetlock, and ringbone. For the relief of the two latter double plantar neurectomy had already been tried after firing had failed, but the result was not a success. The conclusion arrived at by another veterinary surgeon (who was in charge of the case) and myself was that the lameness was probably almost entirely due to the mechanical interference caused by the exostosis at the fetlock joint; but, as there was a great desire to save the mare if possible, and there was decided tenderness over the region of the splints, median neurectomy was performed in order to alleviate this. Immediately after the operation there was indeed a perceptible improvement, but not sufficient to enable her to be sent to any work. The animal did not improve further, and has since been sold and lost sight of.

CASE 66.—Light van gelding, eight years old, very lame owing to the presence of splints on the inside of the cannon bone. Ringbones were also present. The animal had been lame for nine months, and had been fired and blistered on both places twice.

2nd March 1897. Median neurectomy was performed by request of the veterinary surgeon in charge of the case without any immediate improvement, but at the end of a fortnight the animal gradually got sound and was sent to work about 12th April. Since then it has been working regularly, and has shown no return of the lameness.

CASE 67.—Hunter gelding, nine years old, acutely lame on the off fore leg. The animal could not bear to place any weight upon it, and went on three legs. This condition of lameness had been present for about six months, but for the past two years the patient had suffered from intermittent lameness (particularly marked after a hard day). There was a splint, exceedingly tender to pressure, extending underneath the tendons just below the knee; the tendons in this region were also slightly thickened and painful, and there was a small ringbone. The latter had been fired and blistered without permanent improvement being shown. After consultation with the veterinary surgeon in charge of the case, lameness was diagnosed as being due to pain produced by the pressure of the plantar nerve between the splint and the tendon.

20th March 1897. Median neurectomy was performed under chloroform. Immediately after the operation the animal trotted well, using the leg freely and only showing a lameness that would be perceptible to an expert.

21st April. The wound had healed well, but there was still perceptible lameness after work.

26th June 1897. I saw the animal again, and found that there was now a distinct lameness; the animal had carried its owner through a cavalry training, but had shown lameness after duty. When warm and at work the lameness was not noticeable. After another consultation we decided to perform subcutaneous periosteotomy (and insert a seton) over the splint, and at the same time perform external plantar neurectomy. There was no immediate benefit, but about three weeks later there was no lameness at all, and since then the animal has passed through another cavalry training successfully.

CASE 68.—Light cart mare, aged, had been in present owner's possession about ten days and lame all the time. The animal had a very large splint (which appeared to be the cause of lameness) under the tendons; the latter were chronically thickened and tender on pressure in the region of the splint, and there was also a ringbone. Actual cautery had been applied at some time or other to the splint.

3rd April 1897. Median neurectomy under chloroform; there was a perceptible improvement after the operation. The wound healed well, and the animal recovered sufficiently to do slow work, in which it is at present employed.

CASE 69.—Van gelding, aged, had been very lame off and on for about twelve months, and was at present very lame. The animal has a ringbone, an exostosis on the inside of the fetlock, and thickened and contracted tendons. Double plantar neurectomy was performed about three weeks ago, in order to relieve the pain caused by the ringbone, but the result was unsuccessful.

6th April 1897. Median neurectomy was performed as a last resource. Immediately after the operation the lameness was so slight as to be scarcely noticeable, and on 3rd May even this had passed off completely, so that the animal was sent to work.

4th June. Animal worked well until a week ago; is now very lame on the same leg, the fetlock being swollen. Gelatinous degeneration is evidently taking place. Since that date I have been unable to trace the case, but I expect that the animal has been slaughtered.

CASE 70.—Cob, nine or ten years, was lame when bought about thirteen weeks ago; suffering from large exostoses on the inside and outside of the fetlock, the joint appearing almost anchylosed; there was also a thickened tendon. The animal had previously been fired, and had also had double plantar neurectomy performed without benefit. The case seemed a hopeless one, but, as a last resource, and thinking that perhaps the chronic tendinitis might account for a great deal of the lameness, median neurectomy was performed (13th April 1897). After the operation there was a decided improvement, and the animal looked like being fit for walking work.

4th May. Still further improvement, and the animal eventually worked well at walking work for some time. The horse has since been sold, and I have been unable to trace it.

CASE 71.—Cab gelding, suffering from lameness due to neuromas at the seat of operation of a double plantar neurectomy which had been performed six months before; is now far too lame for work.

15th April 1897. Median neurectomy under cocaine. Lameness scarcely perceptible after the operation; the wound healed well, the remaining lameness passed off, and the animal has worked regularly ever since.

CASE 72.—Trap mare, about nine years old, very lame on the off fore leg, due to chronic tendinitis of the perforans and perforatus tendons. She had been lame for six months, and had been blistered without permanent benefit. This was the case operated upon before the West of England Veterinary Medical Association.

22nd April 1897. Median neurectomy under the anæsthetic influence of a mixture of eucaine and cocaine. Immediately after the operation the mare showed no lameness when trotted, and continued to progress favourably for a fortnight, after which time, Mr Nelder informs me, she again became lame. At the present time Mr Nelder tells me that she is still not quite sound, but is sufficiently so to be at work in a butcher's cart occasionally.

CASE 73.—Cab mare, seven years old, has been very lame for three months. The lameness was diagnosed as being due to a large chain splint; there was also a ringbone present. About three weeks previously subcutaneous periosteotomy had been performed on the splint, and the ringbone had been fired, but without success, as the animal was still much too lame for work.

30th April 1897. Median neurectomy under cocaine. Immediately after the operation the lameness was very perceptibly diminished. On 14th May the animal was still too lame for work, but eventually it got all right, and was worked for a while, after which it was sold and lost sight of.

CASE 74.—Van mare, aged, suffering from lameness due to an exostosis under the knee and ringbone. The animal had been lame for fifteen weeks, and had been fired on the ringbone without any improvement resulting.

3rd May 1897. Median neurectomy under cocaine. After the operation there was only slight lameness when trotting. The wound healed well, and the animal has been working regularly ever since. Occasionally she shows slight lameness, owing to a corn on the inside of the foot of the same leg, but when this is pared out the lameness passes off.

Remarks.—This case is of particular interest, as showing that a large amount of sensation is left in the structures on the inside of the foot.

CASE 75.—Cab mare, seven years, has been lame four weeks; very lame from a large exostosis on the inside of the cannon bone and ringbone.

11th May 1897. Median neurectomy under cocaine; there was no improvement after the operation.

17th May. Very lame; external plantar neurectomy was performed, but there was no improvement.

25th May. The animal was still very lame, and the owner was advised to have it destroyed.

CASE 76.—Cab gelding, aged, had been lame off and on for eight months; has a large splint and an exostosis on the inside of the fetlock joint; had been fired on the fetlock without improvement, and was now dead lame.

5th May 1897. Median neurectomy under cocaine; immediately after the operation no lameness could be detected. When the patient, returned on the 13th it was very lame again, and the owner stated that it had met with an accident to the fetlock (which was swollen) and then fallen acutely lame. On the 19th the animal was still very lame, and the owner decided to have it destroyed.

CASE 77.—Cab mare, aged, had been lame two months. The lameness was diagnosed as being due to chronic tendinitis; a ringbone was also present.

17th May 1897. Median neurotomy under cocaine. Unfortunately the student who was operating, pricked one of the posterior radial veins and got a lot of hæmorrhage in consequence; when the nerve was cut the lower end was lost and could not be recovered (in order to remove a portion) without enlarging the wound; on account of the hæmorrhage we were unwilling to do this. There was no lameness after the operation.

25th May. A large pocket of pus had formed, the leg being very much swollen and very sore. A seton was inserted in order to provide drainage, and removed on the 14th of June. The animal went to work on the 21st of June, and has been working regularly ever since.

CASE 78.—Cab mare, nine or ten years old, has been lame on and off for two years. The lameness was due to chronic tendinitis and an exostosis inside the fetlock joint.

18th May 1897. Median neurectomy under chloroform. There was severe lameness before the operation, but afterwards none could be detected except by an expert. The wound healed well; the animal has been turned out to grass ever since, and is not at all lame.

CASE 79.—Cab mare, aged, had been very lame on both fore legs (especially the off) for about a month. On the off leg there were exostoses on the inside of the cannon bone, slightly thickened tendons and a ringbone; on the near fore leg there were exostoses on the lower part of the cannon bone and a ringbone.

3rd June 1897. Median neurectomy was performed on both fore legs under chloroform. There was only a very slight lameness on the off fore after the operation. On the 25th of June this lameness was so slight as to be perceptible only to an expert, and the animal was sent to work. The owner worked the animal for a month, until financial difficulties caused it to be sold, since which time I have been unable to trace it further.

CASE 80.—Pony mare, aged, very lame on both fore legs, particularly the near one. There was a large ringbone on each leg, and a large splint under the knee of the fore.

19th July 1897. The animal was chloroformed, double plantar neurectomy being performed on the off fore leg, and median and external plantar neurectomies on the near fore. There was no lameness after the operation. The wounds healed well and the animal has been at regular work ever since.

CASE 81.—Cart mare, six or seven years old, has been lame for some months; is suffering from chronically sprained and thickened tendons, splint, and ringbone. Actual cautery had been employed, but without beneficial result, about four months ago by the veterinary surgeon attending the case, and it was by his request that the operation was performed on the 20th July 1897. There was no immediate improvement perceptible.

4th August. The animal walks better but still trots lame; external plantar neurectomy was suggested on account of the ringbone, but not consented to. The animal is decidedly improved and works well at walking work, although still showing lameness if trotted.

CASE 82.—Pony mare, had been very lame for several weeks; is now suffering from a ringbone and sprain of the check ligament. The ringbone had been fired and blistered four weeks before without benefit.

15th July 1897. Median neurectomy under chloroform. There was no improvement after the operation. A week later, however, the lameness was perceptibly less, and eventually it passed off. The animal has been in regular work ever since.

CASE 83.—Cab gelding, seven years old, had been in the present owner's possession eight months, and had been lame on and off all the time, sometimes on one fore leg and sometimes the other. On the near fore there was a splint about as large as a tangerine orange extending under the tendons, and an exostosis at the bottom of the radius. On the off fore there were splints on the outside and inside as large as walnuts. The animal was now lame on both fore, being worse on the near.

22nd July 1897. Median neurectomy on both fore legs. The animal was too much under the influence of chloroform afterwards to be able to trot. The wounds healed well, and the animal has been working well ever since without any return of the intermittent lameness.

CASE 84.—Cab mare, aged, has been lame off and on for ten weeks, and very lame for ten days or so. The lameness was diagnosed as being due to a large splint under the knee; the animal has also a ringbone.

6th August 1897. Median neurectomy under chloroform. Unfortunately, during the progress of the operation, the student operating pricked the posterior radial artery and vein. These were ligatured and the wound sutured. There was a great improvement noticeable after the operation. Since then the case has progressed well, there is no lameness, and the animal is back at regular work again.

CASE 85.—Cab mare, six or seven years old, very lame from large exostoses on the inside and outside of the near fore leg.

In July subcutaneous periosteotomy was performed on each of these and setons inserted; these caused an improvement, but there was still severe lameness after the animal had been trotting for a while.

9th August 1897. Median neurectomy under chloroform. There was a decided improvement after the operation. Eventually the wound healed, there was no lameness, and she worked regularly for about three weeks. At the end of three weeks she fell lame on the near hind, and rather than go to the expense of keeping her any longer the owner sold her.

CASE 86.—Cart mare, aged, suffering from a small splint, slight chronic thickening of the tendons, and a large ringbone. The latter had been fired without beneficial result.

3rd September 1897. Median neurectomy under chloroform. There was no lameness after the operation. This case is still under treatment, but promises to make a good recovery.

CASE 87.—Cab gelding, about nine years old, had been in the present owner's possession about seven weeks, and had been lame off and on the whole time; was now very lame. The foot was contracted and had a concave sole (suspicions of navicular disease); there was also a small ringbone, and a very tender chain of small splints on the inside of the cannon bone. The splints had been fired at some time or other previously.

18th September 1897. Median neurectomy was performed, and there was no lameness perceptible after the operation. The case is still under treatment, and promises to make a good recovery.

Summarising the above eighty-seven cases, I think that it will be acknowledged that the results are quite as satisfactory as were those which were published in the Journal last year. Altogether the operation has been performed ninety-seven times, as in ten cases both legs were operated upon. In no case up to the present have I been able to trace a ruptured tendon or sloughing of the hoof unless the external plantar nerve had also been divided. At least thirteen of the cases have now been at regular work for about eighteen months since the operation, whilst ten others have been working for more than twelve months.

I am more firmly convinced than before that for old-standing lameness, where due to splints, exostoses anywhere on the inside of the leg, chronically sprained, thickened, and painful tendons, or cases of that kind which cause pain by pressing on the adjacent nerve structures, after all other treatment has failed, median neurectomy is the operation which will often give the animal a new lease of life and usefulness.

That the whole of the sensation is not removed from the inside of the limb can readily be demonstrated by pricking the skin with a pin, and also by the fact of a corn, when very bad, causing lameness; also by Case 22, in which a wound on the inside of the coronet, and Case 30, in which severe thrush, each caused temporary lameness.

That excision of the median nerve does not affect the healing of wounds in the parts below on the inside, is shown by those cases in which wounds were surgically or accidentally inflicted after the operation.

Case 24, in which a neuroma formed on the median nerve, is worth a special note, as M. Pellerin, in his pamphlet on median neurectomy, draws especial attention to its rarity, as evidenced by the fact that he has never met with or heard of a case.

EDITORIAL ARTICLES.

HEREDITY AND DISEASE.

It could not, without a sacrifice of candour, be said that the discussion on "heredity in its relations to the diseases of animals" at the recent annual meeting of the National Veterinary Association has shed much fresh light on that admittedly obscure subject. It would perhaps have been unreasonable to expect that the occasion would do much to elucidate what may be termed the mechanism of heredity, but one might not unnaturally have looked for some new facts and observations bearing on what is certainly the all-important question in connection with the subject of heredity, viz., What diseases of the lower animals ought to be classed as hereditary?

We cannot help thinking that an undue amount of attention was given both by the essayist and by some of those who took part in the discussion to the ingenious theories that have been put forward to explain the mechanism of heredity, for, as a matter of fact, such of these so-called theories of heredity as have any claim to verisimilitude scarcely serve to make the mechanism of heredity one whit clearer to the ordinary mind. As Professor Dewar has shown in the essay which formed the basis of the discussion at the National Meeting, the cell is by no means the simple nucleated mass of homogeneous protoplasm which Schwann supposed it to be. Both nucleus and cell-substance have a comparatively complex structure, and the act of fertilisation is not a simple case of fusion of a male and a female cell, but the union of definite elements—the chromosomes or idants—of the male spermatozoid with like elements of the female cell or ovum. But admitting this to be true, will anyone say that it dispels, or in any appreciable degree diminishes, the mystery of heredity? The ovum and the spermatozoid have compressed within their microscopic limits the minutest characters of the parent, and in the act of fertilisation these are carried over to the new individual, but the phenomenon is equally mysterious whether we regard the cell-substance or the nuclear chromatin as the vehicle by which the parental characters are transmitted. No theory yet put forward enables us to form a clear conception of how it is that the fertilised ovum of a cow culminates in the production of a new individual of the bovine species, and not in a foal or lamb, and until that, which is the simplest problem of heredity, has been solved we cannot expect to find any clue to the hereditary transmission of morbid characters from a study of the act of fertilisation or the structure of the germ cells.

In the discussion of speculative theories regarding the mechanism of heredity, it appears to be often forgotten that we owe to observation and experiment all the accurate information that we possess concerning the transmission of characters and conditions from parent to offspring. We cannot even distinguish the ovum of a mare from that of a cow, and it is incredible that our microscopic analysis of the ovum or spermatozoid will ever carry us so far as to enable us to discover in them the rudiments of parental defects or morbid characters in process of transmission to the embryo. If splints, or spavins, or side-bones are hereditary, there is assuredly only one way in which the fact can be proved, viz., by patiently collating the results of breeding from animals with these defects.

At the outset of his paper on heredity in relation to disease Professor Dewar has thought it necessary to utter a warning against confusing the terms "hereditary" and "congenital," but he omitted to define one other term regarding which it was equally necessary to avoid misunderstanding, viz., disease. Clearly there can be no useful discussion regarding the rôle of heredity in the production of "disease" between persons who use that word in a different sense. In the absence of a definition on the part of the essayist and the others who took part in the discussion at Reading, we have only the context to guide us as to what they understood by disease, but we note one or two passages which suggest that the speakers were using the word in a new and entirely inadmissible sense. For example, among the list of diseases said to be "undoubtedly hereditary," we find diarrhœa and colic, which makes it evident that it is not disease, but a diminished resistance to the cause of disease, that is here said to be hereditary.

People in the habit of using terms loosely may be content to make one word suffice both for disease and for what is usually termed predisposition, but if utter confusion in the discussion of heredity is to be avoided, a clear distinction must be drawn between the two conditions.

What is disease? There is only one way by which we can define that, viz., by first defining health. Health is that condition which, on examination of the largest available number of individuals, is found to be the common or prevailing one. This applies equally to structure and function. The normal or healthy structure of any individual is that which observation has shown to be the common one among other animals of the same kind, and, in like manner, a condition of health implies that the functions of the various organs are performed as they are in the majority of other animals of the same species. Health being thus defined, we agree to call any departure from it disease. In other words, a part or organ is diseased when in structure or function it departs from the common or prevailing type.

What, it may next be asked, is predisposition? In order to answer that it is necessary to observe that what, according to the above definition, we term disease, is probably never spontaneous. In the immense majority of cases the structural alterations and functional derangements which we regard as disease are the direct effects of agents acting on the part or organ affected, or they represent the reaction of the tissues of the body to such agents. For example, inflammation of the intestinal mucous membrane may be the direct result of, and diarrhœa the reaction to, a poison accidentally administered. Fracture of a bone may be the direct result of mechanical violence, and the formation of a callus the reaction of the bone to the injury. Glanders is the result of the invasion of the body by the germ of glanders, and the glanders lesions for the most part represent the reaction of the invaded tissues to the glanders bacillus. But the resistance offered to direct injury, whether by a blow or a pathogenic organism, varies from individual to individual, and so also does the reaction of the tissues. Where the resistance offered is slight we may say that the individual has a predisposition to that particular disease. Thus, an animal with thin or weak bones is predisposed to fracture, and one whose tissues offer a feeble reaction to the glanders bacillus is predisposed to glanders.

These considerations will serve to show how necessary it is to draw a sharp distinction between predisposition and disease, and in the light of them it becomes apparent that while only a small number of diseases can be said to be strictly inherited, the hereditary transmission of predisposition to disease is likely to be the rule.

Indeed, it appears that one must either altogether deny the transmission of predisposition, or admit that it must be universal in its operation. Countless observations prove that characters which we regard as the most trivial in the individual, such as the quality of the voice, tricks of gesture, and the most minute details of features, may be passed on from parent to offspring, and therefore on *a priori* grounds it would be unreasonable to deny that the qualities of cells and tissues which determine the degree of resistance offered to the causes of disease, are just as likely to be transmitted.

It must be admitted, however, that while the principle of hereditary transmission of predisposition is probably universal in its operation, there are many diseases in which it almost escapes observation. When what may be called the average resistance of the species to the cause of any given disease is very low, then the transmission of varying degrees of predisposition is apt to be lost sight of. On the other hand, where the average resistance of the species to the cause of a disease is high, the occurrence of varying degrees of susceptibility in different individuals forces itself on our attention. For example, the average resistance of animals of the bovine species to cattle plague is slight, and we therefore seldom notice any marked predisposition to that disease in individual cattle; the average resistance offered by the same species to foot-and-mouth disease is considerable, and in almost every outbreak one cannot fail to notice varying degrees of susceptibility in different individuals. Nevertheless, it is probable that predisposition varies from individual to individual just as much in the one case as in the other.

From a practical point of view predisposition is important only when the intensity of the cause of disease, and the degree of resistance offered by the species are so balanced as to bring out distinct degrees of predisposition in different individuals, and even then predisposition to disease ceases to be of practical importance if the cause of the disease can be generally avoided. For example, if, as is frequently supposed, the formation of splints and other exostoses in connection with the bones of the extremities is the result of concussion, there is abundant evidence in favour of varying degrees of predisposition, and since in the ordinary course of a horse's work concussion is unavoidable, it is manifestly indicated to avoid breeding from animals that have developed such bone diseases. Or, to take another illustration, if Professor Dewar is correct in supposing that side-bones are induced by wearing shoes with high calkins, there must be a varying predisposition to side-bones, for every horse shod with high calkins does not develop that abnormality. Hence, it would be indicated to discard for breeding purposes animals affected with side-bones—that is, assuming that their progeny must be shod with high calkins. But although Professor Dewar himself believes that cattle exhibit varying degrees of predisposition to rinderpest, he would hardly recommend that farmers should cease to breed from all animals save those that have an unusual degree of immunity to that disease. In this and in many other cases varying predisposition may be neglected by the breeder, because even when most highly developed the powers of resistance to the cause of the disease are not such as to be practically useful. That is why it would be absurd to disqualify an animal for breeding because it had experienced fracture of a bone or sprain of a tendon, although, by the way, we observe that Professor Dewar includes sprains in his list of hereditary diseases.

As was to be expected, tuberculosis received a good deal of attention at Reading, and we observe with regret that a considerable number of the speakers reaffirmed their belief in the hereditary nature of that disease. For the most part the statements on this subject were mere declarations of faith, unsupported by any arguments, and therefore not calling for refutation. Where any show of reasoning in support of the hereditary nature of tuberculosis was made, it took the form of the oft exposed fallacy of inferring hereditary transmission from the occurrence of tuberculosis in the progeny of tuberculous parents. This is a method of reasoning which is excusable on the part of anyone who denies that tuberculosis may be spread by contact, but it is difficult to understand how it can be seriously advanced by those who admit that in favourable circumstances the disease is contagious.

Much of what was said on the subject of tuberculosis at the recent meeting of the National Association suggests that there are people who regard the retention of beliefs acquired in early life as a sort of virtue. In our view, beliefs and opinions should be held just as long as the available evidence appears to justify them, and no longer. Twenty years ago a belief in the hereditary transmission of tuberculosis was general, and the available knowledge of the period appeared to justify it. Long experience had proved that a far larger proportion of cases of tuberculosis occurred among the progeny of tuberculous parents than among those born of healthy parents, and since Koch's bacillus and the opportunities for its transference from animal to animal by contagion had not then been recognised, it was perfectly natural to suppose that tuberculosis was frequently or generally passed on from parent to offspring at the act of conception. But when it was proved that tuberculosis was due to a specific germ, and experiment had shown that it was possible to infect animals with it at will, it, of course, became necessary to inquire whether infection after birth was not accountable for a large proportion of the cases previously regarded as hereditary. This could obviously be settled by *post-mortem* examination of the newly born progeny of tuberculous parents, and the information derived from that source has made it quite impossible for anyone to maintain that any considérable proportion of animals of the human or bovine species come into the world with evidence of tuberculosis in any of their organs. It is true that those who have pinned their faith to the view that tuberculosis is generally inherited do not admit that the absence of visible lesions from a new-born animal is conclusive evidence of freedom from tuberculosis, for, they say, the bacillus may be there in a state of dormant vitality. To say that this is a perfectly gratuitous assumption is not enough—it is a conclusion entirely opposed to such evidence as we can obtain on the point. Every experiment of the kind that has hitherto been made justifies the assertion that when tubercle bacilli are introduced into the tissues of an animal, whether that animal be newly born or aged, these bacilli either multiply and excite the formation of tuberculous lesions, or they promptly perish. Finally, as bearing on this point, it may be mentioned that tuberculin, which is a more delicate test for the existence of tuberculosis than the most painstaking *post-mortem* examination, has already shown that the progeny of tuberculous cows or bulls are rarely tuberculous at birth.[1]

In taking leave of this question an apology almost seems necessary for having set out once more the reasons for declining to admit that the hereditary transmission of tuberculosis plays a rôle of any importance in the spread of the disease. Foals have been born affected with glanders, and indubitable cases of congenital strangles are on record, but we could not treat seriously the argument that the hereditary transmission of these diseases is of practical importance, or that we ought to take account of it in devising measures of prevention. As we have already hinted, the belief that tuberculosis is generally transmitted by inheritance appears to be an article of faith with those who hold it, and it is not to be upset by reasoning.

In that part of Professor Dewar's essay which treats of heredity in relation to tuberculosis, the author does not give his views regarding the hereditary transmission of the disease, but he does make it clear that he attaches great importance to the hereditary transmission of predisposition as a contributory cause of the present prevalence of tuberculosis among cattle. He says: "There is undoubtedly a predisposition to the disease communicated to the progeny; and the more generations affected with the disease the greater is the predisposition to it, until ultimately they cease to survive to procreate their species."

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[1] Assuming that 20 per cent. of all the cows in Great Britain are tuberculous, and that 1 per cent. of the calves born of tuberculous parents are diseased at birth—an estimate which we believe to be much too high—it would follow that not more than 1 calf in 500 inherits tuberculosis from its parent. And it ought to be pointed out that even this estimate is calculated to convey an exaggerated impression of the rôle which heredity plays in the transmission of tuberculosis among our cattle, since one is apt to overlook the fact that probably only a small proportion of calves tuberculous at their birth reach an age at which they can be used for breeding purposes.

The opposite view has, he says, been maintained in this country by those whom he contemptuously terms the "pure microbiologists."

The passage is a little ambiguous, for it leaves it doubtful whether Professor Dewar means that the "pure microbiologists"—whoever they may be—deny that a predisposition to tuberculosis is transmitted by heredity, or merely refuse their assent to the statement that the predisposition increases with the number of generations affected until the strain is naturally exterminated. If the latter meaning is the one intended to be conveyed, we can only say that we would prefer to enrol ourselves on the side of the pure microbiologists. It is significant that Professor Dewar could not find evidence in support of this view of the deadly nature of tuberculosis nearer than Auvergne. It deserves to be quoted at length:—"Sanson relates at length a case in which a number of Devon cows and bulls, which had been imported, were removed from the Agricultural Institute at Versailles to a model dairy in the mountains of Auvergne, at Saint-Angeau, in the department of Cantal. They were introduced amongst a herd of the finest Auvergnate cows, the purpose being to cross the native cows with the Devon bulls to improve the milking qualities of the animals, but it was a failure. In a few years all the Devons, male and female, died out from tuberculosis, and when Sanson visited the dairy in 1868, the crosses had all died out except one, while the pure Auvergnates remained healthy. These animals were all stabled together, all grazed together on the same pastures, and Sanson contends that if the dissemination of the disease had been entirely due to contagion the Auvergnate cows would also have suffered, whereas all without exception escaped."

It will be observed that while Professor Dewar quotes this extraordinary case as evidence of a growing predisposition in successive generations affected with tuberculosis, Sanson himself obviously interpreted the facts as evidence of the hereditary transmission of the disease itself, for, of course, if there was a great difference of predisposition between the two races, the fact that all the animals of the Auvergne breed escaped, while all the Devons contracted the disease, was in no way opposed to the view that the spread of the disease among the latter was entirely due to contagion. It ought also to be noted that this happened thirty years ago, before Koch's bacillus had been thought of, and before the days of tuberculin. Did Mr Sanson verify the cause of death in every one of the Devons and crosses, and how did he know that none of the Auvergne animals were affected?

We leave it to the clinicians, who, according to Professor Dewar, are all on his side, to say whether it is in conformity with their experience that tuberculosis is such a deadly disease that it will exterminate a herd of Devons or any other breed in the course of a few years. In the very paper from which we are quoting it is said that tuberculosis among cattle is not a very contagious disease, and yet we are asked to believe that in this Auvergne herd it was so contagious that in a few years every Devon and cross-bred animal in the herd became affected ! Our incredulity is not with regard to the number of animals that contracted the disease, but with regard to its invariably fatal course. We would not like to say with Professor Dewar that tuberculosis is not a very contagious disease, but we would without hesitation assert that even among Devons it is not a very fatal one, and that there has not yet been produced any evidence to show that predisposition to tuberculosis is increased by an attack of the disease, or that the reinforced predisposition is transmitted to the next generation.

As to denying the occurrence of predisposition, that is another matter. We never heard of a microbiologist in this or any other country who did not believe that animals of different breeds and families, and even individuals of the same family, exhibit varying degrees of resistance when they become the subjects of tuberculosis, and that the qualities of cells and tissues which underlie this power of resistance are likely to be transmitted by heredity. But, unfortunately, this resistance varies within rather narrow limits, and in the case of our valuable breeds it never amounts to absolute immunity, or even to such a degree of insusceptibility as will enable them to indefinitely withstand the risks of infection when they are housed with other tuberculous animals.

There does not appear to be much cogency in citing the resistance of Auvergne cattle to tuberculosis, unless it is meant to suggest that the owners of our pedigree herds of shorthorns, Devons, or Aberdeen-Angus cattle should discard these, and replace them by some semi-wild or mountain breed possessing more marked powers of resistance to tuberculosis. The problem to be solved is to free our existing herds from the disease without sacrificing those desirable qualities which have been built up as characters of the breed by long-continued selection. We may admit that most of our most valued strains of cattle are readily infected with tuberculosis, but those who own them would much rather keep them with this defect than deteriorate the strain by crossing with an inferior breed absolutely immune against the disease, if such a breed could be found.

We can imagine someone saying that it is not necessary to go outside any of the strains to find animals without this fatal predisposition to tuberculosis, and that by steadily selecting such animals for breeding, and discarding those affected with tuberculosis and all their progeny, it would be possible in the course of a few years to create a herd immune against the disease. It is, of course, not possible to absolutely disprove such a view, but it is easy to show how slender are the foundations on which any such expectations are built. How, in the first place, are we to ascertain which of the animals in any given herd are devoid of predisposition ? Are we to regard present freedom from tuberculosis on the part of an animal as evidence of immunity? The supposition is too absurd, for, as has already been mentioned, the tuberculin test if carried out soon after birth would by this method of reasoning prove that almost all animals of the bovine species are immune. Or are we to take freedom of several successive generations as the only evidence of insusceptibility to tuberculosis? That would be an impracticable proposal, for in most cases we could not obtain the assurance that the progenitors of an animal for even two generations had been free from tuberculosis. In short, it is a great fallacy to assume that even in the same herd it is solely or mainly the varying degrees of predisposition that determine which animals shall become the victims of tuberculosis. As well might it be maintained that it is always the most susceptible horse that contracts glanders, or credit the animals that do not react to mallein in an outbreak of glanders with immunity against that disease. Nor can the predisposition to tuberculosis be detected by a study of conformation, though it appears that there are still to be found clinicians who believe the opposite. Tuberculin has taught us that the narrow chest and other points of conformation, which were once accepted as evidence of a "phthisical constitution," are of no value whatever as indications of special predisposition to tuberculosis. Such characters are the *effects* of tuberculosis, not the predisposing cause of that disease, and the most splendid type of conformation is quite compatible with the usual degree of predisposition to tuberculous infection. That these statements are true will not be denied by anyone who has had much experience in the use of tuberculin as a test for tuberculosis in cattle.

Professor Dewar finds fault with those who say that if the sources of infection are cut off from healthy animals predisposition may be disregarded, and he remarks that "this may be quite true theoretically, but it is not a very practical way of dealing with the matter." To that it may be replied that if a method is correct in theory it cannot be wrong in practice. What is the alternative method? To breed only from animals that are free from tuberculosis, and neglect the risk of contagion! That, it may safely be asserted, is wrong in theory, and it would be disastrous in practice. The only sound advice to give to those who are fortunate enough to possess healthy herds is to warn them not to ascribe their present freedom from tuberculosis to absence of predisposition on the part of their animals, or to neglect any reasonable precaution against infection. In like manner, we are entitled to tell those who own herds already affected that the one condition necessary to get rid of the disease is to make a permanent separation between the diseased and the healthy, and that, no matter what the breed or strain may be, a herd may be made and kept free from tuberculosis.

THE CURE OF TETANUS.

THE series of experiments by Professor Nocard which we publish in this number will be read with interest by those who have been anxious to learn the truth regarding the value of antitetanic serum in the treatment of tetanus, but it is to be feared that they will come as a shock to those who have committed themselves to the opinion that the serum is reliable as a curative in that very intractable disease. It is possible that some of those who have been fortunate enough to see their patients recover after the serum treatment, will still give the new agent the credit of having averted a fatal termination, but unbiassed minds must recognise that Professor Nocard has justified his contention that a curative agent for tetanus has yet to be discovered.[1]

To determine what is the value of the serum method of treatment is a problem that would require a very large body of clinical evidence for its solution. Professor Nocard says that clinical observation is inadequate to resolve it, but that is perhaps going too far. Clinical experience was probably quite competent to settle this question in the long run, just as it has determined the value of quinine in the treatment of malaria, or of mercury in syphilis; or as the clinical experience which Professor Nocard himself appeals to has shown the value of antitetanic serum as a prophylactic. Indeed, one can imagine that the mere record of the results of treatment in a not very large series of naturally occurring cases of tetanus might have established the superiority of the serum over every other method of treatment, in the same way as a comparatively brief experience of iodide of potassium served to convince everyone that it left every other agent far behind in the treatment of actinomycosis. But it was quite obvious from the outset that, whatever was the value of the serum in the treatment of declared tetanus, it had no claim to rank as a specific. The few instances in which it had been tried showed that it could not be relied upon to cure every case; all that could with any plausibility be maintained was that it appeared to conduct to recovery some cases that would otherwise have had a fatal termination, and even that estimate probably overstepped the limits of caution. Had tetanus been as invariably fatal as rabies, the practitioner who saw three recoveries under the serum treatment might have been justified in rushing into print with the cry that a specific had been discovered, but, as we all know, a certain number of cases—and occasionally a considerable proportion of successive cases—terminate in recovery under the most varied treatment, or without any treatment at all, and it argued a very ill-balanced judgment to vaunt antitetanic serum as a curative agent of high value because two out of three horses treated with it had recovered.

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[1] Strong clinical evidence in the same direction is furnished in the most recent issue of the Berliner Thierärztliche Wochenschrift (No. 39, 1897) wherein it stated that, out of nineteen cases of tetanus in the horse treated at the Berlin Veterinary College by intravenous injection of tetanus antitoxin between June and September of this year, sixteen have proved fatal.

In discussing the value of antitetanic serum, it ought to be remembered that that agent is not a substance of fixed strength, and that it is therefore possible, and even probable, that some of the serums in the market are much weaker than the samples used in Professor Nocard's experiments; at any rate, it is very unlikely that the serum now being offered to the practitioner is stronger than that which has entirely failed as a curative when submitted to the rigid test of experimentation. Nor is there much room for hope that serum of greater antitoxic power than that used in M. Nocard's experiments will be obtained by present methods.

In a sense, though not in the chemical one, antitetanic serum is capable of neutralising the tetano-toxin, and of averting the effects of that poison when the two agents act simultaneously on the animal body, but apparently the serum is not a remedy against the *effects* of the toxin, and hence its failure in cases where the toxin has already for several days been exerting its injurious influence on the system. In natural tetanus, occurring as a wound infection, the period of incubation is seldom less than a week, and during the whole of that time the toxin elaborated by the bacilli is steadily exercising its injurious effects. Before the practitioner is called in to treat the case the animal in many instances has already experienced the action of a fatal dose, and intervention is then too late.

The second part of Professor Nocard's article is as encouraging to the use of antitoxic serum as a prophylactic as the first part is damaging to its claims to be considered curative. Here the results of experiment entirely accord with those obtained in practice, and conclusively prove that antitetanic serum is a reliable preventative, when injected before infection, or within a brief period afterwards. Fortunately, there are few districts or places in this country in which tetanus is so prevalent as it appears to be in some localities in France, but when the reliability of an injection of serum to confer a period of protection against the disease becomes generally known, it will doubtless be frequently employed both in human and in veterinary practice. When used with this object its cost is not prohibitive, for a comparatively small dose is all that is required to confer protection.

THE DUTIES OF VETERINARY SURGEONS UNDER THE NEW PUBLIC HEALTH (SCOTLAND) ACT.

THE recently passed Public Health (Scotland) Act contains provisions that must be disappointing to those who, like Mr Ernest Hart, are anxious to see the inspection of milch cows and meat committed to "medical men with special training in cow conditions."

On the other hand, it cannot fail to give satisfaction to the veterinary profession, as it goes farther than any previous Act in recognising the necessity of consulting members of their body in the matter of dairy and meat inspection.

The provisions of the Act that are of special interest to veterinary surgeons are those that relate to the erection of public slaughter-houses, the inspection of dairies, and the inspection of meat.

Section 34 empowers the Local Authority of any district, other than a burgh, to erect slaughter-houses within or without their district, or to combine for that purpose with neighbouring Local Authorities. Although the section is only permissive, one may hope that it will help towards the gradual extinction of the dirty, practically uncontrolled, private slaughter-houses that are still the rule in most parts of Scotland. When public abattoirs take the place of these it may be regarded as certain that they will be supervised by veterinary inspectors.

Section 43 deals with the question of unsound food. It empowers any medical officer, sanitary inspector, or veterinary surgeon approved for the purpose by the Local Authority, to inspect any article of food or any animal intended for food which is exposed for sale, but it stipulates that in the case of any proceeding under the section with regard to a living animal the medical officer or sanitary inspector, unless he is himself a qualified veterinary surgeon, shall be accompanied by an approved veterinary surgeon. The most important clause of this section, however, is that which empowers each Local Authority to appoint a place within its district for the examination of any animal alive or dead, and to appoint a veterinary surgeon whose duty it shall be for a fixed fee to examine such animals, and to pass or condemn the same as regards their fitness for human food. The importance of this lies (1) in the fact that the veterinary surgeon is exclusively recognised as the meat inspector, and (2) in the fact that a preceding part of the section exempts from penalty or imprisonment any person accused of offering for sale unsound meat, provided he can prove that the meat in question had, within a reasonable time prior to the seizure thereof, been examined and passed by a veterinary surgeon approved for the purpose.

Section 60 deals with the inspection of dairies, and it stipulates that the medical officer of health must be accompanied by a veterinary surgeon, approved for the purpose, when he examines animals whose milk is supposed to be causing disease among human beings, and any report by the medical officer of health regarding such an inspection must be accompanied by the report of the veterinary surgeon.

It is evident that the powers committed to members of the veterinary profession by this Act are of a very responsible character, and it is earnestly to be hoped that experience of its working will justify the confidence which the legislature has reposed in them.

Reviews.

Annual Reports of Proceedings under the Diseases of Animals Acts, the Markets and Fairs (weighing of cattle) Acts, etc., for the year 1896. London: Eyre and Spottiswoode.

THIS is, under a changed name, the Annual Report of the Veterinary Department of the Board of Agriculture. It is not quite as much belated as its predecessor, but nevertheless the year to which it relates had been six months gone before it was issued.

In addition to the usual statistical tables relating to disease in home and foreign animals, international trade in animals, etc., it comprises reports by the chief veterinary officer, and the Principal of the Animals' Division. The former extends to twenty-two pages, and while it deals with the incidence of all the scheduled contagious diseases of animals that occurred in Great Britain in 1896, it is mainly devoted to swine-fever, and the diseases which may or have been confounded with that. An interesting account is given of the history, symptoms, etiology, and morbid anatomy of swine-fever, and two of the rarer lesions of the disease are excellently depicted in coloured lithographic plates.

The report by the Principal of the Animals' Division deals with the same diseases from an administrative point of view, and is accompanied by two useful maps showing the distribution of swine-fever and rabies in Great Britain in 1896. The price of the Report is only 10d., and every veterinary inspector ought to procure it.

A Critical Period in the Development of the Horse. By J. C. Ewart, M.D., F.R.S., Regius Professor of Natural History, Edinburgh. London: Adam and Charles Black, 1897.

PROFESSOR EWART, whose recent researches on the development of the horse are well known to readers of this Journal, has utilised the material which he had collected for the purpose of these researches in an endeavour to find an explanation of the proneness of many mares to break service from the sixth to the ninth week. His examination of young equine embryos has shown that about the end of the seventh week, just before the villi begin to sprout out from the allantois, the attachment between the embryonic sac and the uterus is apt to be less secure than at any other period of pregnancy. This anatomical peculiarity, and possibly a tendency to early extrusion of the embryo inherited from marsupial ancestors, are suggested as important factors in determining abortion in the mare.

The monograph deals with a subject of great interest and importance to horse-breeders and veterinary surgeons, and it may be recommended on the additional ground that it contains the fullest and most accurate account of the development of the equine fœtal envelopes that has yet been published.

CLINICAL ARTICLES.

CONTAGIOUS INFLAMMATION OF THE LUNGS AND PLEURA OF YOUNG CATTLE.

By JOHN PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

UNDER the head of Septic Pleuro-pneumonia, several continental writers have described a fatal disease affecting calves during the first three months of their lives. Poels[1] gives the results of his observations on the affection in Holland, and refers to its existence in the province of Antwerp. Perroncito[2] has written a full description of an infectious pneumonia of the calf which caused great losses in Italy. In British text-books I find no mention of the occurrence in this country, though Youatt,[3] writing before the scheduled pleuro-pneumonia of cattle is believed to have been introduced, has a chapter in his book on "Cattle" headed, Acute and Epidemic pneumonia, which is in some points suggestive. Crookshank[4] refers to a septic pleuro-pneumonia affecting calves up to two months old, the course being rapid, and death occurring on the second or third day. This writer does not mention its existence in this country, but, like Poels, states that it is communicable and fatal to rabbits, guinea-pigs, and mice.

I have within the past few years met with an affection which bears some resemblance to the descriptions alluded to above, one of the most remarkable differences being that the age of its subjects in the outbreaks investigated by me ranged from six months to two years.

In one instance a farmer purchased three calves about nine months old, of which one was noticed ailing on its arrival home, and died on the following evening. The other two were now placed in a byre, and on the next day one became very ill, and it succumbed on the fourth day after its arrival. The third showed symptoms of the affection on the same day as the last named, became extremely debilitated, and on the fifth day was destroyed as hopeless. Only the viscera of these animals were seen *post-mortem* by a veterinary surgeon. In the course of two months after this, the young stock reared on the farm, whose ages ranged from six to eighteen months, were turned into the same byre. On the seventh day following four animals were noticed to be very ill, and one died during the night. A veterinary surgeon was sent for, who found three calves giving evidence of pleurisy and pneumonia of an acute character. He then took the temperature of the remaining eleven, and finding in three that it was elevated (104°-105°), removed those whose temperature was normal to another situation. On the following morning pulmonary symptoms were manifest, and of the six, four died within four days. The other two survived, but continued to do badly for a considerable time. *Post-mortem* revealed pleurisy and pneumonia of the character about to be described.

In the second instance a veterinary surgeon was called in to see nine calves from six to ten months old, which were supposed to be suffering from "husk," being informed on his arrival that three had died on the day previous. The nine calves were coughing, some having a more or less profuse discharge from the nostrils and eyes, while auscultation and percussion revealed the existence of pleurisy and pneumonia, and there was the grunting which so commonly accompanies these affections in cattle. Depression was well marked and increased rapidly. The patients sometimes, though not invariably, remained standing until the later stages, when they persistently lay down. The temperatures ranged from 104° to 106°. In two of these cases diarrhœa existed. Of the nine, four died, and five made a slow recovery, making a total loss of seven out of twelve attacked. In each of the fatal cases, which occurred in the younger calves, death took place within the third day after the first symptoms were manifest. These calves had never been turned to grass, but were fed on a patent calf food, hay, crushed oats, and chaff. On the same farm were twenty-nine other young cattle above twelve months old, which had the run of an adjoining byre separated by railings. Of these, several became affected in a milder form, but all recovered, though a cough remained for many weeks; and notwithstanding a generous diet they continued in poor condition for a long time. *Post-mortem* examination revealed conditions almost identical with those of the former outbreak.

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[1] "Fortschritte der Med." 1886.
[2] "Il Med. Vet." 1885.
[3] "Cattle" 1837.
[4] "Bacteriology and Infective Diseases," p. 227.

In another instance twenty animals were affected, the majority dying within ten days, and showing similar appearances *post-mortem*, but with more broken down gangrenous tissue.

Under the head of "Septic Pneumonia in Calves," Mr James Smith of Huntingdon read a paper to the Lincolnshire Veterinary Medical Association in June 1892. At the time Mr Smith consulted me and submitted some specimens of lung. This was a very severe outbreak, and many of the lesions were in a state of gangrene. On this farm, at the time of Mr Smith's first visit, six animals were found by him dead, and ten very ill. This was the third outbreak which had occurred on the place within three years. From the writer's statements and those of other members who joined in the discussion, it was evident that losses from this disease had been attributed to "husk," scheduled pleuro-pneumonia, and tuberculosis. The disease was most successfully arrested by means then suggested on the lines to be found at the end of this paper. It is worthy of remark here, that in all cases, including the above, which have come under my notice, the proprietor had been in the habit of purchasing young stock.

In some of the instances referred to, the after-death appearances have so simulated those of the scheduled pleuro-pneumonia of cattle, that lungs have been submitted to my examination under some suspicion of the existence of that disease; and though in most of the descriptions of so-called septic pleuro-pneumonia of calves, gastro-intestinal lesions are spoken of, I have met with such once only in the affection now referred to. In this case diarrhœa existed, and probably depended on parasites in the gastro-intestinal tract. The constancy of the morbid changes appears to prove the disease to be essentially one of the lungs and pleura, though the pericardium is not infrequently involved. In one case only have I observed the whole of both lungs implicated. Usually there has been what is sometimes known as patchy pneumonia of one or both lungs, the pleura corresponding being generally involved. The anterior lobes have generally been affected in the cases examined by me. The affected lobules, usually darker in colour, are well marked out by the septa infiltrated with lymph, giving a marbled appearance resembling that so familiar in the contagious pleuro-pneumonia of cattle. The altered lung tissue, however, was less firm ; and the interlobular bands were narrower, contained much less liquid, and were more easily broken down. The affected pleura was less thickened and darker in colour. The foci of disease were more numerous, and manifested a greater tendency to break down and become gangrenous than in the scheduled affection. The bronchi in some cases contained a considerable amount.

My experience leads me to the conclusion that this affection is contagious, that the virus remains effective for a considerable time outside the animal body, and contaminates the places in which affected calves are kept, thus accounting for the recurrence of the affection as an enzootic. There is evidence of a short period of incubation, and the course in fatal cases is rarely more than three days. Calves under twelve months old become more readily and seriously affected than those above this age.

Though in advanced acute cases curative treatment is not hopeful, a large measure of success has followed the means suggested. Separation of diseased from the healthy by aid of the thermometer, removal of both lots to a fresh situation after all have been washed about the eyes, nostrils, chest, and feet with a solution of some suitable disinfectant, such as carbolic acid, are among the first procedures. The diseased have been then subjected to inhalation of burning tar, carbolic acid, sulphur, or eucalyptus, strict attention being paid to the hygiene of the animals, nursing, etc.

Intratracheal injection of carbolic acid was tried on four animals, which died shortly afterwards.

The place inhabited by the affected should be thoroughly disinfected.

CONTAGIOUS OPHTHALMIA IN CATTLE.

By JOHN PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

IN the summer of 1892, I was consulted in the matter of several calves, about six months old, which had suddenly become affected with inflammation of the eyes, leading to temporary blindness. Of thirteen animals kept in a shippen, ten had, without appreciable cause, developed acute inflammation of the cornea and conjunctiva. At first three were noticed to move about strangely, and on examination the cornea was found to be opaque. In the course of a week seven others had become similarly affected. The three remaining unaffected were then removed to a paddock, and showed no signs of the disease. Up to this time, there being no evidence of contagion, the mischief was attributed to mechanical or chemical irritation connected with food stuffs. It happened, however, that a cow which was in a yard into which the ten calves were turned for treatment developed the affection. This naturally excited some suspicion of contagion.

During the summer of 1895, two similar occurrences were reported to me from different parts of the country.

In July 1896 I was consulted by Mr L. Barrett, M.R.C.V.S., of Romford, Essex, regarding an outbreak of the disease in a herd of about fifty young cattle. Here, in the first instance, four animals were found affected, and in course of a fortnight forty were suffering. By a rigorous treatment the disease was for a time limited to this lot of young stock, but in about a month some of the cows on the farm, which were being attended by men who had attended the calves, became affected, and also another lot of calves. Through an unfortunate mistake in a telegram, I was unable to make a direct experiment in order to prove whether it was contagious. During the past summer, however, an opportunity occurred under similar circumstances. Some of the discharge was collected from the conjunctivæ of two affected animals, and inserted between the eyelids of two healthy cattle in another situation and under different conditions. Both young animals developed the affection, and in ten days one of two healthy calves placed with these contracted it.

The symptoms shown are those usually exhibited in inflammation of these parts. Photophobia, redness of the conjunctiva, more or less suffusion of the eyelids, muco-purulent discharge, opacity of the cornea, and in many cases ulceration. In the larger proportion of cases both eyes were affected. In severe cases the temperature has been raised a degree or two, but beyond the consequences immediately depending on the local changes there was little constitutional disturbance. In about 3 per cent. there has been permanent opacity of one or both corneæ.

Treatment which appeared successful consisted in separating the apparently healthy from those yielding evidence of the disease. The affected were placed in a darkened shed, and the eyes of both lots were carefully and thoroughly sponged with solution of sulphate or chloride of zinc, borate of soda, weak nitrate of silver, or some such collyrium twice daily for two or three days. After which to the affected eyes boracic or nitrate of mercury ointment diluted with vaseline as occasion required were applied. Care should be taken in disinfecting sponges, etc., also the buildings in which the animals have resided.

OUTBREAK OF CŒNUROSIS, GID, OR STURDY IN LAMBS.

By JOHN PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

SPORADIC cases, depending on the presence of the cœnurus cerebralis in the cranial cavity of sheep, are among the commonest occurrences in the flocks of Great Britain. It is amongst lambs that the more severe losses are occasioned by this parasite; indeed, amongst adult sheep the affection is generally regarded as rare. The symptoms shown and the life history of the cestode on which they depend are so well known that it would be superfluous to refer to them in detail here. There may be, however, some points of interest connected with the following outbreak.

The flock consisted of about 900 lambs, some of which began to show cerebral symptoms early in May last, and from that time to the present between sixty and seventy have died from "gid," *post-mortem* examination revealing in all cases one or more of the hydatids in the brain. Notwithstanding, it was hard for the owner to reconcile such great losses with the presence of the parasite which is usually found affecting sheep singly, or but few at a time. The lambs had been very highly fed, and on certain changes, from tares, etc., to pasture, being made, the disease appeared to lessen, and the owner was disposed to attribute the fatalities to errors of diet. Notwithstanding the assurances of his ordinary professional adviser, I was consulted under the circumstances. Examination left no doubt of the correctness of diagnosis of the veterinary surgeon in attendance. Cœnuri were found in varying numbers and in almost every part of the brain. As is usual in young lambs, the period between the first manifestation of symptoms and death was much shorter than that obtaining in older sheep. It often happened that the lambs were found dead four days after being noticed at all abnormal. Though the cœnuri were found in various parts of the brain, the power to stand was usually lost in a day or two, when the animal fell on its side and succumbed within four days, during which, in the majority of cases, the familiar movements of the limbs were more or less constantly performed. Previous to falling, the symptoms were various according to the situation, size, etc., of the cyst; the turning in a circle was comparatively rare, and the holding back of the head and trotting motion of the fore limbs were most commonly obtained. Stupor and general paralysis affected a few, but, in no instance was there observed any bulging of the frontal bones.

In connection with this outbreak, whose fatalities I fear cannot yet be counted, the more remarkable features appear to me to be the extent of the disease, the exclusive affection of lambs (not a single ewe has been so far affected), and that 147 lambs brought into this flock from another farm have remained perfectly healthy. Many lambs under three months old have been fatally affected. It is admitted by some continental writers[1] that the embryos may pass from the mother to the fœtus, which, is therefore affected at birth. There is in my opinion considerable room for doubt concerning such an admission, to which the freedom of the ewes from disease in this case lends no support.

On the estate in question there are a large number of dogs kept for sporting purposes and working sheep. It is thus not far to seek for the host of the tapeworm, of which the lamb is the intermediary bearer. Though there is not much hope attaching to any curative measures at our disposal, by administration of vermifuges to the dogs, destruction of their fæces, etc., and by preventing them from feeding on the heads of affected sheep, there is every reason for believing that the life cycle of the parasite may be broken and a recurrence prevented.

I am specially induced to report the above outbreak, as the course of the affection in lambs is so rapid that it is liable to be mistaken. It often happens that the symptoms ordinarily attributed to gid are overlooked by the shepherd, and the veterinary surgeon has only the opportunity of seeing a patient on its side struggling and unable to stand, when it is by no means easy to locate the cause of the trouble.

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[1] Friedberger et Fröhner, Pathologie, etc., tom. ii., 79.

Abstract and Reports.

PUBLIC HEALTH (SCOTLAND) ACT, 1897.

THE following are the clauses of this Act which relate to slaughter-houses and the inspection of unsound food and dairies by veterinary surgeons.

34. The Local Authority of any district other than a burgh may provide, establish, improve, or extend and maintain within or without their district, and two or more such Local Authorities may combine to so provide, establish, improve, or extend and maintain fit shambles or slaughter-houses for the purpose of slaughtering cattle, and for that purpose may borrow such sums of money as they shall find necessary on the security of the public health general assessment, and of the rates to be taken and levied for the use of such shambles or slaughter-houses and ground on which the same are erected, or on any one or more thereof, and the provisions of section one hundred and forty-one of this Act shall, with the necessary modifications, apply to such borrowing.

43. *Unsound Food.*—(1.) Any medical officer or sanitary inspector or any veterinary surgeon approved for the purposes of this section by the Local Authority may at all reasonable times enter any premises within the district of the Local Authority, or search any cart or vehicle, or any barrow, basket, sack, bag, or parcel, in order to inspect and examine and may inspect and examine

(*a*) any animal, alive or dead, intended for the food of man which is exposed for sale, or deposited in any place or is in course of transmission for the purpose of sale or of preparation for sale; and

(*b*) any article, whether solid or liquid, intended for the food of man, and sold or exposed for sale, or deposited in any place or in course of transmission for the purpose of sale or of preparation for sale,

the proof that the same was not exposed or deposited or in course of transmission for any such purpose, or was not intended for the food of man, resting with the person charged; and if any such animal or article appears to such medical officer or sanitary inspector or veterinary surgeon to be diseased, or unsound, or unfit for the food of man, he may seize and carry away the same himself or by an assistant, in order to have the same dealt with summarily by a sheriff, magistrate, or justice.

Provided that in the case of any proceeding under this section with regard to a living animal the medical officer or sanitary inspector, unless he is himself a qualified veterinary surgeon, shall be accompanied by a veterinary surgeon approved as aforesaid.

The police force of each police area shall have power to search carts or vehicles, or barrows, baskets, sacks, bags, or parcels, and to assist generally in executing and enforcing this section.

(2.) If it appears to a sheriff, magistrate, or justice, that any animal or article which has been seized or is liable to be seized under this section is diseased, or unsound, or unfit for the food of man, he shall condemn the same, and order it to be destroyed or so disposed of as to prevent it from being exposed for sale or used for the food of man; and the person to whom the same belongs or did belong at the time of sale or exposure for sale, or deposit or transmission for the purpose of sale, or of preparation for sale, or in whose possession or on whose premises the same was found, shall be liable to a penalty not exceeding fifty pounds for every animal or article, or if the article consists of fruit, vegetables, corn, bread, or flour, for every parcel thereof so condemned, unless he proves that he and the person acting on his behalf (if any) did not know, and could not with reasonable care have known, that it was in such a condition, or where the proceedings are before a sheriff, at the discretion of the court, if it finds that he has knowingly and wilfully committed the offence, he shall be liable without the infliction of a penalty, to imprisonment for a term of not more than three months with or without hard labour, and also to pay all expenses caused by the seizure, detention, or disposal thereof.

Provided that if such person proves that the animal or part thereof condemned as aforesaid was within a reasonable time prior to the seizure thereof examined upon the premises where the animal was slaughtered and passed by a veterinary surgeon approved as aforesaid called in for the purpose, and who shall have granted a certificate of passing as nearly as may be as in the next subsection provided, or by a veterinary surgeon in terms of that subsection, he shall be exempt from penalty or imprisonment under this section for such offence.

(3.) Each Local Authority, or two or more Local Authorities in combination, may, if they think fit, appoint a place or places within its district or their districts, and fix a time or times at which a veterinary surgeon approved as aforesaid shall attend for the purpose of examining any animal alive or dead which may there be submitted to him, and passing or condemning the same, and such veterinary surgeon shall, on receipt of a fee to be fixed by the Local Authority or Authorities and paid by the owner, examine and pass or condemn in whole or in part any animal or carcase so submitted to him; and if he shall pass the same he shall grant a certificate of passing which shall set forth the name of the owner, the date and hour of examination, and such particulars regarding the animal or carcase as the Local Authority or Authorities may prescribe for the purpose of aiding in the subsequent identification of the same; and if he shall condemn the animal or carcase, or part thereof, the animal or carcase or part so condemned shall be retained and be forthwith destroyed by the Local Authority or Authorities or so disposed of as to prevent it from being exposed for sale or used for the food of man, and the owner shall be entitled to the net price realised from the residual product of the carcase or part so condemned, if any, after deducting the expenses of condemnation and destruction. Provided that no carcase shall be submitted for examination, either under this or the immediately preceding subsection, unless as a whole carcase, including the thoracic and abdominal viscera, in such manner that the examiner shall be readily able to satisfy himself that the organs are those of the carcase under inspection.

(4.) Where it is shown that any animal or article liable to be seized under this section and found in the possession of any person was purchased by him or consigned to him from another person for the food of man, and when so purchased or consigned was in such a condition as to be liable to be seized and condemned under this section, the person who so sold or consigned the same shall be liable to be brought to trial in the district in which such animal or article was seized, and on conviction shall be liable to the penalty and imprisonment above mentioned, unless he proves that, at the time he sold or consigned the said animal or article, he and the person acting on his behalf, if any, did not know, and could not with reasonable care have known, that it was in such a condition.

(5.) A copy of any certificate granted by a veterinary surgeon, under subsections two or three of this section, shall forthwith be sent by him to the chief constable of the jurisdiction in which the examination of the animal or carcase took place, and the certificate itself shall be sent by the person selling the animal or carcase forthwith after the sale, and not more than seven days from the date of the certificate, to the chief constable of the jurisdiction in which the sale of the animal or carcase took place, and if any veterinary surgeon or person shall contravene this enactment he shall be liable to a penalty not exceeding twenty pounds.

(6.) Where a person convicted of an offence under this section has been within twelve months previously convicted of an offence under this section, the sheriff, magistrate, or justice may, if he thinks fit, and finds that the offender knowingly and wilfully committed both such offences, order that a notice of the facts be affixed, in such form and manner and for such period not exceeding twenty-one days as the sheriff, magistrate, or justice may order, to any premises occupied by that person, and that the person do pay the costs of such affixing, and if any person obstructs the affixing of such notice, or removes, defaces, or conceals the notice while affixed during the said period, he shall for each offence be liable to a penalty not exceeding five pounds.

(7.) If the occupier of a licensed slaughter-house is convicted of an offence under this section the sheriff, magistrate, or justice convicting him may cancel the licence for such slaughter-house.

(8.) If any person obstructs a medical officer, sanitary inspector, or veterinary surgeon as aforesaid in the performance of his duty under this section he shall, where the proceedings are before a sheriff, and where the sheriff is satisfied that the obstruction was with intent to prevent the discovery of an offence under this section, or that the accused has within twelve months previously been convicted of such obstruction, be liable to imprisonment for any term not exceeding one month in lieu of any penalty authorised by this Act for such obstruction.

(9.) A sheriff, magistrate, or justice, may act in adjudicating on an offender under this section whether he has or has not acted in ordering the animal or article to be destroyed or disposed of.

60. (1.) If the medical officer of any district has evidence that any person in the district is suffering from an infectious disease attributable to milk supplied within the district from any dairy situate within the district, or that the milk from any such dairy is likely to cause any such disease to any person residing in the district, such medical officer shall visit such dairy, and the medical officer shall examine the dairy and every person engaged in the service thereof or resident upon the premises or who may be resident in any premises where any person employed in such dairy may reside, and if accompanied by a veterinary surgeon approved as aforesaid, shall examine the animals therein, and the medical officer shall forthwith report the results of his examination accompanied by the report of the veterinary surgeon, if any, to the Local Authority or any committee of the Local Authority appointed under section 14 to deal with such matters.

(2.) If the medical officer of any district has evidence that any person in the district is suffering from any infectious disease attributable to milk from any dairy without the district, or that the milk from any such dairy is likely to cause any such disease to any person residing in the district, such medical officer shall forthwith intimate the same to the Local Authority of the district in which such dairy is situate, and such other Local Authority shall be bound, forthwith, by its medical officer to examine the dairy and the persons aforesaid, and by a veterinary surgeon approved as aforesaid, to examine the animals therein, previous notice of the time of such examination having been given to the Local Authority of the first-mentioned district, in order that the medical officer or any veterinary surgeon approved as aforesaid may, if they so desire, be present at the examinations referred to, and the medical officer of the second-mentioned Local Authority shall forthwith report the results of his examination, accompanied by the report of the veterinary surgeon, if any, to that Local Authority or any committee of that Local Authority appointed under section 14 of this Act to deal with such matters.

(3.) The Local Authority of the district in which the dairy is situated, or any committee appointed for the purpose, shall meet forthwith and consider the reports together with any other evidence that may be submitted by parties concerned, and shall either make an order requiring the dairyman not to supply any milk from the dairy until the order has been withdrawn by the Local Authority, or resolve that no such order is necessary.

(4.) Where proceedings are taken or any order is made under this section by the Local Authority of a district other than a burgh, it shall not be competent to appeal against the said proceedings or against said order of the county council.

(5.) The Local Authority may, if the dairy is within the district, require the dairyman not to supply milk either within or without the district, and shall give notice of the fact to the Local Authority of any district within which they believe milk to be supplied from such dairy.

(6.) Any such order shall be forthwith withdrawn on the Local Authority, or their medical officer on their behalf, being satisfied that the milk from the dairy is no longer likely to cause infectious disease.

(7.) It shall be open to any Local Authority or dairyman aggrieved by any such resolution or order, or withdrawal of order, to appeal in a summary manner to a sheriff having jurisdiction in the district in which the dairy is situated, and the sheriff may either make an order requiring the dairyman to cease from supplying milk, or may vary or rescind any order which has been made by the Local Authority, and he may at any time withdraw any order made under this section. Pending the disposal of any such appeal the order shall remain in force.

(8.) If any person refuses to permit the medical officer or veterinary surgeon of either Local Authority to make examination as above provided, or, after any order has been made under this section, supplies milk in contravention of the order, he shall be liable to a penalty not exceeding ten pounds, and if the offence continues, to a further penalty not exceeding five pounds for every day during which the offence continues.

(9.) Provided that—

(*a*) proceedings in respect of the offence shall be taken before a sheriff having jurisdiction in the place where the dairy is situate; and

(*b*) a dairyman shall not be liable to an action for breach of contract if the breach be due to an order under this section.

(10.) Nothing in or done under this section shall interfere with the operation or effect of the Contagious Diseases (Animals) Acts, 1878 to 1886, or of any order, licence, or act of the Privy Council or the Board thereunder, or of any regulation, licence, or act of a Local Authority, made, granted, or done under any such order of the Privy Council or the Board, or exempt any dairy, building, or thing, or any person from the provisions of any general Act relating to dairies, milk, or animals.

Whenever it shall be certified to the Local Authority, by the medical officer, or other legally qualified medical practitioner, that the outbreak or spread of infectious disease within the district is, in the opinion of such medical officer or medical practitioner, attributable to milk supplied to any dairyman, whether wholesale or retail, or to milk supplied by one or other of several such dairymen, whether wholesale or retail,—

(1). The Local Authority may require such dairyman, whether within or without the district, to furnish to them within a time to be fixed by them being not less than twenty-four hours, a full and complete list of names and addresses of all his customers within the district so far as known to him, and such dairyman shall furnish such list accordingly, and the Local Authority shall pay to him for every such list at the rate of sixpence for every twenty-five names contained therein; and every person who shall wilfully or knowingly offend against this enactment shall for each such offence be liable to a penalty not exceeding five pounds and to a daily penalty not exceeding forty shillings;

(2.) The Local Authority may require such dairyman to furnish to them, within a time to be fixed by them, a full and complete list of the names and addresses of the farmers, dairymen, or other parties from whom, during a period to be specified, the milk, or any part of the milk which they sell or distribute, was obtained, and, if required, to produce and exhibit to the medical officer, or to any person deputed by him, all invoices, pass-books, accounts, or contracts, connected with the consignment or purchase of milk during such period, and such dairyman or others shall furnish such lists and produce and exhibit such invoices, pass-books, accounts, or contracts, accordingly; and every person who shall wilfully or knowingly offend against this enactment shall for every such offence be liable to a penalty not exceeding five pounds and to a daily penalty not exceeding forty shillings;

(3.) In any case where the person liable to any penalty under this section is not resident within the district, such penalty may be sued for at the instance of the Procurator Fiscal before the sheriff of the county in which such person is either domiciled or carries on business.

ROYAL VETERINARY COLLEGE, LONDON.

LIST OF BURSARIES, MEDALS, HONOUR CERTIFICATES, ETC., 1896-97.

Coleman Prizes.

Silver Medal	J. F. Thurston.
Bronze Medal	S. B. Woollatt.
Certificate of Merit	F. C. Gillard.

Centenary Prizes.

Class A	R. J. Collings.
Class B	W. Lawson.
Class C	T. Wolsey.
Class D	J. F. Thurston.

CLASS PRIZES.

Class D.

MEDICINE.—*Silver Medal*—J. F. Thurston. *Bronze Medal*—A. F. Harber. *First-Class Honour Certificates*—S. T. Amos, J. B. Collyer, P. M. Evershed, A. J. Hines, R. W. Hall, J. R. Jackson, G. H. Kitchen, P. Lloyd, A. Miller, C. E. Perry, W. J. Watt, F. T. Walder, S. B. Woollatt, B. E. Wooster, R. Wood. *Second-Class Honour Certificates*—F. C. Gillard, A. Olver.

SURGERY.—*Silver Medal*—J. R. Jackson—*Bronze Medal*—F. T. Walder. *First-Class Honour Certificates*—A. F. Harber, C. E. Perry, J. T. Thurston, S. B. Woollatt. *Second-Class Honour Certificates*—S. T. Amos, J. B. Collyer, P. M. Evershed, F. C. Gillard, A. J. Hines, R. W. Hall, G. H. Kitchen, P. Lloyd, A. Miller, A. Olver, W. G. Taylor, W. J. Watt, B. E. Wooster, R. Wood.

Class C.

MATERIA MEDICA.—*Silver Medal*—E. Peacey. *Bronze · Medal* — C. Radway. *First-Class Honour Certificates*—W. Anderson, C. W. Abrams, G. W. Bloxsom, J. Chalmers, A. H. Cory, W. Cranston, D. Chalmers, T. Elliot, R. Hudson, J. W. Oakley, P. J. Simpson, H. A. Woodruff, T. Wolsey, W. R. Williams. *Second-Class Honour Certificates*—E. A. Batt, E. Brown, R. W. Carless, D. Crole, H. Gamble, C. F. Parsons, P. W. Smith, A. Whicher.

HYGIENE.—*Silver Medal*—T. Wolsey. *Bronze Medal*—C. Radway. *First-Class Honour Certificates*—C. W. Abrams, E. Brown, W. Cranston, D. Crole, D. Chalmers, E. Peacey, H. W. Percy. *Second-Class Honour Certificates*—W. H Anderson, E. A. Batt, G. W. Bloxsom, R. W. Carless, A. H. Cory, O. S. Fisher, H. Gamble, A. Hudson, R. Hudson, C. F. Parsons, P. W. Smith, C. M. Sharpe, P. J. Simpson, H. A. Woodruff, W. R. Williams, A. Whicher.

PATHOLOGY.—*Silver Medal*—T. Wolsey. *Bronze Medal*—H. A. Woodruff. *First-Class Honour Certificates*—E. Peacey, C. Radway. *Second-Class Honour Certificates*—W. Anderson, C. W. Abrams, E. A. Batt, G. W. Bloxsom, R. W. Carless, A. H. Cory, W. Cranston, D. Crole, T. Elliott, R. Hudson, C. F. Parsons, C. M. Sharpe, P. J. Simpson, W. R. Williams, A. Whicher.

Class B.

ANATOMY.—*Silver Medal*—G. Lockwood. *Bronze Medal*—W. Lawson. *Second-Class Honour Certificates*—H. G. Allen, H. Burrell, J. H. Poles, C. E. Rix, G. H. Wooldridge.

HISTOLOGY.—*Silver Medal*—G. Lockwood and C. E. Rix (æq.). *Bronze Medal*—W. Lawson. *First-Class Honour Certificates*—H. G. Allen, A. M. Brodie, H. Burrell, C. Roberts, E. L. Siddall, J. M. Tate, G. H. Wooldridge. *Second-Class Honour Certificates* — C. R. Copp, C. H. Coombe, J. W. Flanagan, B. L. Lake, G. V. M'Naboe, J. C. Munby, H. Mosley, W. R. Neale, J. H. Poles, A. L. Purdy, J. P. Stableford, G. Sykes.

PHYSIOLOGY.—*Silver Medal*—W. Lawson. *Bronze Medal*—C. E. Rix. *Second-Class Honour Certificates*—H. G. Allen, A. M. Brodie, H. Burrell, G. Lockwood, J. Lee, W. R. Neale, J. H. Poles, A. L. Purdy, J. M. Tate, G. H. Wooldridge.

Class A.

CHEMISTRY AND TOXICOLOGY.—*Silver Medal*—H. Greenfield. *Bronze Medal*—R. J. Collings. *First-Class Honour Certificates*—J. Nicholas, J. Share-Jones, H. Thackeray, C. Tyler, C. W. Wilson. *Second-Class Honour Certificates*—H. H. Aldred, H. H. Barker, G. H. Broad, P. T. B. Basset, E. Child, H. Cooper, J. A. Dixon, W. G. Flanagan, E. S. Gillett, K. Hewlett, H. Howard-Jones, A. Leaning, G. May, W. N. Rowston, H. A. Reid, W. S. Stevens, H. L. Simons, H. Sanderson, C. E. Turvill, E. J. Wadley, H. Whitemore, S. J. Young.

PRACTICAL CHEMISTRY.—*Silver Medal*—R. J. Collings. *Bronze Medal*—H. Cooper.

BIOLOGY.—*Silver Medal*—H. Greenfield. *Bronze Medal*—R. J. Collings. *First-Class Honour Certificates*—J. T. Share-Jones, H. Thackeray, E. J. Wadley, C. W. Wilson. *Second-Class Honour Certificates*—H. H. Aldred, G. H. Broad, E. Child, H. Cooper, J. A. Dixon, E. S. Gillett, K. Hewlett, G. D. Horsey, H. A. Lash, A. Leaning, R. E. Mosedale, J. Nicholas, W. N. Rowston, W. S. Stevens, C. Tyler, C. E. Turville, G. Tucker, H. Whitemore.

MINOR ANATOMY.—*Silver Medal*—J. T. Share-Jones. *Bronze Medal*—R. J. Collings. *First-Class Honour Certificate*—H. Thackeray. *Second-Class Honour Certificates*—H. H. Aldred, E. Child, H. Cooper, J. A. Dixon, H. Greenfield, K. Hewlett, J. Nicholas, C. Tyler, G. Tucker, E. J. Wadley.

PASS LIST.

The following are the Pass Lists[1] of this Institution for Session 1896-97.

FIRST PROFESSIONAL EXAMINATION.

Messrs †P. A. Aitchison, *J. W. Flanagan, C. G. Heard, F. E. Mason, H. S. Moseley, R. H. Pinching, †C. E. Steel, *A. Spicer, H. Anthony, T. G. Bush, G. H. Broad, P. F. B. Bassett, H. Cooper, J. A. Dixon, E. S. Gillett, *K. Hewlett, A. Leaning, J. Nicholas, H. A. Reid, J. T. Share-Jones, *H. Thackeray, G. Tucker, E. J. Wadley, H. H. Aldred, H. H. Barker, P. V. Beatty, E. Child, *R. J. Collings, W. G. Flanagan, *H. Greenfield, G. D. Horsey, E. Nicholson, W. N. Rowston, W. S. Stevens, H. Sanderson, C. Tyler, L. M. Verney, C. M. Wilson, *Kumar Shri Jethiji.

SECOND PROFESSIONAL EXAMINATION.

Messrs G. F. Gould, M. Glasse, A. M. Dowling, H. S. Elphick, H. W. Fernandes, R. G. Bell, P. Broad, F. H. Ingersoll, C. Joliffe, F. S. Probyn, *H. P. Sarjeant, R. C. Baxter, H. Burrell, D. S. Dickman, *G. Lockwood, *J. Lee, *J. H. Poles, *C. G. Rix, *D. H. Rait, E. L. Siddall, J. J. D. Tapley, *G. H. Wooldridge, A. M. Brodie, R. L. Green, J. Cane, H. H. Jeffries, W. Lawson, *J. Neale, A. L. Purdie, *C. Roberts, J. P. Stapleforth, *R. Stokoe, *J. M. Tate, E. A. Wilshaw, *H. G. Allen.

THIRD PROFESSIONAL EXAMINATION (Four Years' Course).

Messrs W. H. Brown, J. Buckinghan, *R. C. Cochrane, A. S. Head, F. C' Hobbs, L. W. Heelis, H. T. O'Neill, *C. M. Park, C. W. B. Sikes, J. B' Walker, W. M. Williams, *W. H. Anderson, G. W. Bloxsom, R. W. Carless' W. Granston, T. Elliott, H. Gamble, E. Peacey, C. F. Parsons, P. W. D' Smith, H. A. Woodruff, W. R. Williams, C. W. Abrams, E. Brown, A. H. Cory, D. Chalmers, E. T. Goodall, G. P. Knott, H. W. Percey, *C. Radway, P. J. Simpson, T. Wolsey, A. Whicher, E. T. C. Enory, T. W. Oakley.

FINAL EXAMINATION.

Messrs G. Condor, Bognor; G. H. Butcher, London; S. de M. Woodward, Loughborough; W. M. Power, Waterford; J. Eadie, Rutherglen: H. W. Young, Chingford, Essex; E. T. Stanley, London; F. R. Brandt, Ashstead, Surrey; W. C. Lowe, Merthyr, Tydvil; W. S. Mulvey, London; H. Mitton, Ilkley; C. H. Stephens, Bombay; S. T. Amos, Clevedon; P. M. Evershed, Billinghurst, Sussex; A. F. Harber, Cheltenham; R. W. Hall, Barnstaple; J. R. Jackson, Wadebridge, Cornwall; A. Olver, Washaway, R.S.O.; J. T. Thurston, Tressingfield, Suffolk; F. T. Walder, Crawley, Sussex; B. E. Wooster, Bledlow, Bucks; P. E. Hoyland, Rotherham; F. M. Skues, Nunhead; S. Dixon, London; J. A. Hewson, Ardfert, Co. Kerry; J. T. Jewell, Maidstone, Kent; J. B. Collyer, Weston-super-mare; F. C. Gillard, Grantham, Lincs.; A. J. Hines, Fenton, Staffs.; A. Miller, Cambridge; C. E. Perry, Bristol; W. J. Watt, Ilda, Fairfax, Co. Virginia, W.S.A.: S. B. Woollatt, Ware, Herts; R. Wood, Waterford; W. V. Allen, Downpatrick; F. B. Gresham, Newark, Notts.; R. Hudson, E. Retford, Notts.; C. D. Stewart, Cheltenham; M. Allan, Downpatrick.

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[1] In this and the succeeding Pass Lists † indicates with First-Class Honours, and * with Second-Class Honours.

ROYAL (DICK) VETERINARY COLLEGE, EDINBURGH.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1896-97.

FIRST PROFESSIONAL EXAMINATION.

Messrs J. L. Still, W. J. Boyd, J. Cockroft, F. H. W. Cundell, C. A. W. Cunningham, R. A. Edwards, J. Forrest, G. Garden, T. Harper, W. Hepburn, J. Hobday, †R. B. Littler, T. Nunan, E. A. Ryan, S. E. Sampson.

SECOND PROFESSIONAL EXAMINATION.

Messrs R. R. Arthur, P. Donovan, R. Garret, R. Bryden, A. E. A. Charles, †F. W. Coates, J. Cowan, W. P. Cushnahan, D. G. W. Galt, *J. J. Garside, *R. Garraway, †W. Girvan, †A. Gofton, T. Hogg, C. P. Hynes, J. F. Joyce, †H. W. Leech, *G. H. Livesay, †E. Little, *W. Ludgate, E. Mossman, J. A. Macgregor, †J. McIlvenna, J. J. O'Connor, E. O'Neil, *A. Robb, J. A. Russel, †W. S. King.

THIRD PROFESSIONAL EXAMINATION (Four Years' Course).

Messrs R. W. Hadfield, J. B. Kay, J. H. Brown, A. C. Burton, A. Gibson, T. Loughran, C. A. Reid, H. Ryan, W. Tart, T. H. Tranter, S. A. Winkup, E. Lander, H. Leyshon.

FINAL EXAMINATION.

Messers W. Hunter, W. Newman, ‡W. D. Rees, R. F. Wall, H. O. Thompson, W. T. Bolton, F. Richardson, E. J. Bolton, M. Bray, W. H. Campsall, *J. Brownless, W. Cathcart, †H. Lomas, W. Murgatroyd, J. F. Talbot, J. G. Blackwood, B. S. Cockerton, S. S. Forster, J. Grant, J. H. Norris, W. B. Stableforth, R. C. Smith, T. Wilkinson, A. Scott, J. R. Wardrope, C. Goundry, F. J. Pringle, H. J. A. Bradbury, F. M'Ghee, H. K. Tasker.

GLASGOW VETERINARY COLLEGE.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1896-97.

FIRST PROFESSIONAL EXAMINATION.

Messrs C. Bell, F. P. Beck, T. Price, W. G. Forbes, J. Allan.

SECOND PROFESSIONAL EXAMINATION.

Messrs R. Spittal, D Forwell, *J. Murphy, *R. Porteous, J. P. Small, *J. M'Leod, *W. W. Lang, *A. K. Murray, E. Soutter, W. Watson, D. M'Hattie, J. Montgomery, J. Aitken, F. Timing.

THIRD PROFESSIONAL EXAMINATION.

Messrs W. Crawford, A. Fraser, J. Crawford, R. H. Johnson, J. C. Erskine, D. Weir, J. W. Conchie, H. A. Oliver, J. Smith, R. G. Jones.

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‡ **Entitled to Register and receive his Diploma, 29th March 1897.**

Final Examination.

Messrs H. Ferriers, H. G. Simpson, G. Thompson, H. M'Vean, W. T. Blane, A. S. Brooksbank, W. Mackenzie, *J. F. M'Eachran, J. R. M'Call, G. N. Waugh, G. Broom, F. Russell, J. Rankine, J. Laird, D. M'Geoch.

NEW VETERINARY COLLEGE, EDINBURGH.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1896-97

First Professional Examination.

Messrs T. Parker, F. Moon, E. Ashworth, S. C. Binks, W. G. Brindred, F. Crossley, S. P. Dunphy, H. Martin, †B. Runciman, T. Runciman, A. Spruell, J. Stafford, A. A. Walker, W. H. Bradley.

Second Professional Examination.

Messrs S. Robson, H. Whipp, G. A. Harrison, *J. Paul, J. Kelly, G. E. Hamilton, T. C. Howatson, J. Bradley, †H. Dyson, *J. Evans, *W Gasgoyne, *A. M'Nae, *D. M'Donald, R. A. Plunkett, †H. Taylor, C. R. Levist, R. Butler.

Third Professional Examination.

Messrs R. J. Bailey, *H. H. Fergusson, *A. M. Porteus, *M. Webb, W. M. Ogilvy, E. Morrison, G. J. Roberts.

Final Examination.

Messrs A. P. Beesley, A. W. Findlay, J. Knowles, H. Locke, J. M. Walker, W. L. Jones, T. M. Ryan, W. H. Savage, H. Briggs, H. Brittain, J. Pollard, G. C. Barber, J. H. Lomas, R. S. Rowlands, A. A. Wilson, E. Segar, A. Munro, W. F. Hughes.

ROYAL VETERINARY COLLEGE, LONDON.

INAUGURATION OF THE WINTER SESSION, 1897-98.

The Winter Session at the College was opened on Friday the 1st October, the inaugural address being delivered by Mr D'Arcy Power, M.B., F.R.C.S. The Chair was taken by Colonel Sir Howard Vincent, C.B., M.P., who was supported on his right by the Right Honourable Walter Long, M.P., President of the Board of Agricultural, and Professor G. T. Brown, C.B., and on his left by Colonel Sir Nigel Kingscote, K.C.B., Veterinary-Colonel Lambert, C.B., and Veterinary-Colonel Duck, C.B., Director-General of the Army Veterinary Department. The large lecture theatre was filled to overflowing with an audience comprising the College Staff, members of the profession, and students.

The Chairman, on rising to open the proceedings, said:—

"Mr President and Gentlemen,—I have the honour of attending here to-day, on behalf of the Board of Governors, to welcome you to this new Session of the Royal Veterinary College, and I am quite sure that it is the earnest wish of His Royal Highness, the Duke of Cambridge, and the whole Court of Governors, that the coming session shall be not less successful than all the sessions which have preceded it (cheers). We have the pleasure of seeing here to-day one of the most distinguished members of Her Majesty's Government, viz., the Right Honourable Walter Long, Minister of Agriculture, who is on my right (cheers), and he has attended here to show his great interest in the Royal Veterinary College, and his appreciation of the great services which the veterinary profession is rendering to the country. We have also here many former students who have come to show the great interest which they continue to take in the Royal Veterinary College, and I think they will tell those students who have already been in the College, and especially those students who are entering the College for the first time to-day, that the veterinary profession throughout the country is rising every year in public estimation. The testimony of Mr Long himself will probably tell you that the tendency of agriculture at this time is to greatly increase the pastoral area, and therefore much to increase the number of animals in the country. That being so, the treatment of animals and their freedom from disease and sickness are matters that will demand the greatest care and attention of all who are interested in the welfare of the country, and in the dumb creation.

"It is not my object or purpose to detain you at any length, but I desire to express to the professors and all connected with the College the recognition by the Court of Governors of the services they are rendering. The extension of the curriculum from three sessions to four sessions was at first attended by some misgivings on the part of the well-wishers of the Institution, but now, with some four years' experience, we are able to say that it is a great success, and it increases the confidence of the country in the students of the Royal Veterinary College. In no other place is it possible for them to derive more advantageous instruction in the veterinary art than that which is imparted at the Royal Veterinary College (cheers). It is not only in the theory of the veterinary art that great progress has lately been made, but it is also in the practical work. I am quite sure if there is anyone here who has not been over the premises of the College, they will, on doing so, appreciate the great opportunities for clinical instruction which exist here, and all will form the opinion that everything is being done to make the education at the Royal Veterinary College the very best that can possibly be given (applause).

"It only remains for me to congratulate the veterinary profession on the recognition by Parliament of the increased status they have attained in the country. In the last session the Public Health (Scotland) Act introduced a very important change. Hitherto, the inspection of meat has been left rather indiscriminately to the policeman, the butcher, and the sanitary inspector, but this Act has given a distinct recognition to the veterinary profession, and has done much to place them in the position they ought to occupy in connection with the inspection of animals required for food purposes.

"I congratulate the profession, one and all on the important change that has been made, and I hope that the Royal Veterinary College now opening its 108th session will extend the area of its operations, and that the recognition of the noble veterinary profession will become more and more marked by Parliament, and by the country generally (cheers).

"I will now call on Mr D'Arcy Power, who has been so long connected with the College as a lecturer, and whose name is so favourably known in surgery generally, to deliver the opening address, and I do not doubt that from that address, all, including former and present students, will derive the greatest profit and instruction.

"After Mr D'Arcy Power has delivered the inaugural address, I shall have the honour of calling upon Mr Walter Long, the Minister of Agriculture, to address the College students" (applause).

Mr D'Arcy Power, who, when rising, was greeted with loud cheers, then delivered the following address :—

"Sir Howard Vincent and Gentlemen,—My duty resolves itself into two parts. The first, an easy and pleasant duty. I have to welcome here those who have just joined the ranks of your profession by attaching themselves to our ancient College and who come here to-day for the first time. The second part of my task is no less pleasant, but it is much less easy. I am to send away the seniors in the veterinary profession with that good conceit of themselves which is so necessary a factor towards a sustained advance in every walk of life.

"Let me offer then to those new students who are here to-day for the first time a few words of most hearty welcome. Veterinary science at the present moment is advancing more rapidly than any other branch of the healing art. It will continue to advance, and it will soon attain a high position in the eyes of the State and of private individuals, as one of the most useful professions, both to the wealth of the country and the welfare of the community.

"Much has been done by the veterinary profession within the last few years, much more still remains to be done. The students who now enter here will have to bear the heat and burden of the day, for it will be their duty to aid that advance, and to raise veterinary science to a still higher position. I not only welcome you therefore, but I wish you all good fortune in the career upon which you are now entering. Be sober, be honest, strive to be gentlemen in thought as well as in deed. Since I first came amongst you as a teacher, only seven years ago, you have made enormous advances. You work harder, you write much better papers, you are more attentive to what is told you, and therefore you go to your examinations much better prepared. But there is still great room for improvement. You want a higher standard of general knowledge and a better preliminary examination. You must trust more to your own work. You must think for yourselves more, and have a less abiding faith in 'cram.' Mere 'cram,' leading to a semblance of knowledge, wont help you. Steady work for even a short time daily throughout each session is of far greater avail than feverish all-night sittings beginning a fortnight before the examinations.

"Then when you have obtained your diplomas, try so to conduct yourselves as to become the friend and trusted adviser of your clients. Make them look up to you as to men who hold their position by virtue of real knowledge. Try to be above your clients rather than below them or upon an equality with them. Do not become a mere cattle doctor, but remain as you began a veterinary surgeon. Individual exceptions there must be in every profession, but let these exceptions be of the lower type, and let the rank and file of the veterinary profession show themselves constantly in favour of progress, both in science and in social position. Do not think that your own efforts are useless, or that it cannot matter what example you set. Some of my best friends in the country I am glad to reckon amongst the farmers and stock-owners, and when I visit them I often ask about the veterinary surgeons in the neighbourhood. Formerly I only received a frank confession of ignorance or a doubtful shake of the head; now more often than not I am told he is an excellent useful fellow, and that my friends often call him in, whereas in the more benighted parts of the country they either employed the white witch, or they trusted to the knowledge which had become traditional in their family, and which was often hardly distinguishable from witchcraft.

"And now let me come to the real subject of my address to-day, the debt that human medicine in all its branches owes to veterinary science. The debt is surely a great one and if we take the subjects in the order in which they are taught in the College, we shall find that our founder had a share in beginning it.

"Charles Sainbel taught human anatomy a lesson, and at the same time established his own reputation in England, when he published his essay on the proportions of Eclipse, the great race horse belonging to Mr O'Kelly, who died 27th February 1789, and was never beaten. The essay is still in advance of its time, for it shows how animal mechanics can be expressed in geometrical terms, and it would be interesting to apply Sainbel's methods to the individual record breakers, who are now so numerous amongst us in the world of athletics. The observations of veterinary anatomists, too, explain many obscure points in connection with the development and anatomy of man, especially in those aberrant conditions which are found to a greater or less degree in every body which is carefully dissected.

"The debt of human physiology to veterinary physiology is well recognised. In modern times, and in this country, it dates from the day when Archdeacon Hales, in 1727, introduced a long tube into the femoral artery of a horse, and noted the height to which the blood rose in it. The importance of breeding has led our veterinary surgeons to a most careful observation of the generative functions in animals, and has thus enriched physiology with many facts of prime importance. It has demonstrated the possibility of artificial impregnation. It has settled the vexed question of superfecundation, whilst the recent work on epizootic abortion has set us thinking whether a similar condition may not occur sometimes in the human species.

"But there is no need for me to dwell upon this subject of the debt of human to veterinary physiology, for we have amongst us one of the most distinguished of those to whom the debt is owing. Veterinary-Major Smith is well known to all of us here, and in our minds he bids fair to rank with Meade, Flourens, Colin, and those other great names which are familiar to everyone, as pioneers in a most difficult region of scientific enquiry.

"In like manner too, in histology, the careful examination of animal tissues, and a knowledge of the minute differences occurring in the structure of different animals, is frequently of great service in a study of human tissues. It is often the only way in which to elucidate the effects of disease in man. But, in human histology we have almost everything to learn, for, until the last year or two, all our knowledge has been derived from a study of animal structures, or from diseased or imperfectly preserved human tissues.

"In botany, veterinary science has long caused medicine to place itself under a debt of gratitude. All practical men, whether physicians or veterinary surgeons, have had occasion to study poisonous plants, and their observations upon animals have been of signal service to the medical profession. Veterinary science, however, has done more than this, for it has compelled botanists to study the most difficult but important natural order of the grasses, not only from a mere classificatory standpoint, but in such a manner as to indicate the economic value of its constituent genera.

"Veterinary medicine sometimes gives valuable hints to the human physician about the use of drugs and the cause or the treatment of various diseases, which though they are common to man and animals are more common in animals. Thus, the effects of iodide of potassium in the treatment of actinomycosis can be much more easily observed by veterinary surgeons than by physicians, whilst we have learned from veterinary surgeons the use of eserin in treating some forms of intestinal obstruction. In other cases again, the ætiology of the disease is assisted, and we may thus obtain a clue to its prophylaxis, as in the case of the recent long distance rides in Germany, where horses, healthy at starting, died of pneumonia soon after they had arrived at their destination. Sometimes, too, the observations of veterinary surgeons serve to refute a commonly assigned cause of a disease in the human being. Thus, diabetes was long ascribed to mental worry, yet it is not a very uncommon disease in dogs, and thus a rather persistent vulgar error is exploded by the help of veterinary science.

"Until quite recently, however, the importance of veterinary science to physicians has never been better shown than in connection with the life history of the more common entozoa, some of which use our bodies as an intermediate host in the cycle of their existence, which they renew in cattle, dogs, or rodents. Need I do more here than allude to the work of Professor Spencer Cobbold, who was formerly a Professor at this College? Work which was so remarkable, so accurate and so complete, that with Davaine in France and Kuechenmeister and Leuckart in Germany, the subject ended, for it seemed as if the four great helminthologists had learned all that was to be known about the parasitic worms inhabiting the intestines and the tissues. It is true that they did not study exhaustively the hæmatoza, for the microscope and histological methods were not then sufficiently advanced to enable them to do so, but this only shows that Art is long though Time is fleeting. We are glad to think that one of the greatest of the giant helminthologists was once a teacher in this College, and that his work has thrown a flood of light upon many of the obscure points in connection with the life history of parasites, which has afforded information of equal value to the physician and to the veterinary surgeon.

"The surgeon has learnt somewhat less from the veterinarian than any of his brethren who practice upon the human body, yet even he has learned a few principles from veterinary surgery which have stood him good stead in the advancement of his art. The operations of the sow-gelder stimulated the early ovariotomists to practise a similar operation upon women, and encouraged them to persevere in spite of the most disastrous results, until Sir Spencer Wells so perfected his technique as to lead the operation from a position of complete disrepute to the high place in the estimation of surgeons which it now occupies.

"Indeed it has always been in principle rather than in detail that medicine has been indebted to veterinary science. Nowhere is this better seen than in pathology, which is not only the common foundation upon which human medicine and human surgery are built, but which forms, as we are now beginning to learn, the platform for all treatment, whether of animals or of human beings. The truth of this proposition needs no demonstration at my hands or in this place. You acknowledged it a few years ago when you appointed to the headship of this College one of the best pathologists in Europe, Professor M'Fadyean, who now so worthily presides over it as your Principal. And not only here, but throughout the world, wherever you find a first-rate pathologist you will find that he is not merely a human pathologist but that he is a man of science, a comparative pathologist with a wide knowledge of animals as well as of men. This, I believe, is what has made pathology the grandly progressive science which it has become. John Hunter was interested alike in men and cattle, but his successors for many years were mere morbid anatomists concerned with the details of morbid processes, not with their principles. It is only lately that we have outgrown this stage, and have become pathologists in the true sense, as disciples of Hunter, who must always remain our great master.

"In pathology human medicine has learned much from veterinarians; even morbid anatomy has been helped by the careful work of those who taught us anew the great lesson known long since to the Preacher, but long since forgotten: "For that which befalleth the sons of man befalleth beasts: even one thing befalleth them: as the one dieth so dieth the other: yea they have all one breath: so that a man hath no pre-eminence above a beast."

"The better and wider knowledge of pathology possessed by the best veterinary surgeons is sometimes brought home to medical men in a very practical manner. It happens from time to time, at one of the great pathological societies in this country, that some recondite point of comparative pathology is being feebly discussed by the light of imperfect knowledge derived from one-sided observation, until the ill-concealed anxiety of some of my friends, whom I see here to-day, can no longer be repressed. At length the opportunity arrives, Professor Brown, Axe, M'Fadyean, or Penberthy rises, and I at once settle myself and feel happy. They draw upon stores of knowledge which are quite beyond the reach of all but the very best medical men, and in a moment, in the twinkling of an eye, all is made clear, and pathology has vindicated itself against mere morbid anatomy. It is no wonder therefore that comparative pathology rises daily higher in the estimation of the medical profession, for to the thoughtful physician its problems appear identical with those he is most often called on to solve. Daily too we see that prophylaxis is the most important part of medicine, and it is exactly in the study of prophylaxis that comparative pathology excels, though it has done good service in pointing out the part played by animals in sharing diseases which affect man. In doing so, it has shown that animals may act as transmitters of disease, so that cats and pigeons in diphtheria, horses in influenza, and rats in the plague, must be considered as probable rather than as possible carriers of infection.

"But the question of prophylaxis is essentially one which has taken its rise on the veterinary side of the profession, for it is one with which veterinary science is particularly fitted to deal.

"It will occur at once to everyone that we owe to veterinary science the germ of that great discovery which bids fair to render this century as epoch-making in medicine as it is in steam, electricity, and engineering. The cattle doctors and the cowkeepers in Gloucestershire noticed that those who had been infected with cow-pox were in some sort protected from small-pox. The tradition was long kept amongst themselves, but, almost by accident, it became known at last to Edward Jenner when he was a surgeon's apprentice at Sodbury. The perseverance and the energy of Jenner placed the tradition upon a sound basis of scientific evidence, and upon this pedestal it was allowed to remain isolated for many years. At last, in the fulness of time, came Pasteur, who, with the logic characteristic of the nation he adorned, generalised from the particular, and introduced into pathology, and so into all medicine, the doctrine of the artificial attenuation of virus in infectious disease, a doctrine which has already been modified by the theory of immunity.

"The enterprise of the veterinary profession and of the large stockowners throughout the world has been thoroughly awakened by the results obtained in animals treated by the methods devised at the Institut Pasteur. The value of the successive new discoveries has been tested upon the most extensive scale. The advantages as well as the limits of such remedies as mallein and tuberculin are now duly appreciated. Their use has familiarised a very conservative body of men with the most modern pathological methods, and by so doing has rendered them capable of understanding, and eager to take advantage of, any further advance along the same lines.

"Such a training and such knowledge have enabled several great economic advances to be made within the last few years, though some of the advances are more marked abroad than at home. Thus horse flesh has been added to the staple of food, a rigorous examination of slaughtered pigs has almost eliminated trichinised flesh, for no great outbreak of trichinosis has occurred of late years either here or on the continent of Europe. On the other hand, the health of the community has been improved and the lives of many children have been saved, for doubt has been cast upon the wisdom of eating tuberculous meat, or of drinking milk from tuberculous cows or even from those who have suppurative diseases of the udder, though the animal be otherwise healthy. A knowledge of such facts has led the Medical Officer of Health into very close alliance with the veterinary surgeon, which, I venture to predict, will become still closer as time progresses.

"Prophylaxis is of as much importance to a large stockowner as it is to the headmaster of a public school, and a thorough knowledge of preventive measures is even more valuable to a veterinary surgeon than it is to a physician. Only in exceptional cases will an owner consent to pay for the treatment of a prolonged illness in his stock, whereas a human being must be attended throughout his sickness, for he cannot be slaughtered summarily.

"But all preventive measures on a large scale require special knowledge and concerted measures. Special knowledge only accumulates slowly, concerted measures require the intervention of the State.

"After many years the government in this country was made to feel the necessity of enforcing the application of sanitary measures, and medical officers of health came into existence. In a tentative manner, and very incompletely, some attempts have been made to produce a veterinary sanitary service. Too much is left to the initiative of local bodies, and what has hitherto been done in detail now requires to be established upon a broad groundwork. Such a service, properly conducted, would be of undoubted use to the community, whilst it would be advantageous to the veterinary profession. It would give the profession a status which it does not yet possess, and it would provide within its ranks a more highly educated, perhaps I should rather say, a more highly specialised, class of thoroughly scientific men.

"The subject is too vast to dwell upon in the short space of time now available even were I competent to treat it in detail. The rudiments of such a service already exist, as you well know, but I think that it might be much better organised, much better worked, and much better paid. The duties of the veterinary inspectors at the present time appear to be of far too limited a nature. They are confined to carrying out the provisions of certain Acts of Parliament for the prevention of contagious diseases occurring in animals. I would have it far otherwise. I would place the whole of animal hygiene under the care of a veterinary sanitary service, and, as no one can be much in advance of his time, such a service for the present should be carried out along the lines which have been found most useful in the medical sanitary service, care being taken to eliminate, or if they cannot be altogether eliminated, to strengthen, those points which have seemed weakest in the working of the Public Health Acts.

"Such a veterinary sanitary service would not be hard to organise. Its existence is already foreshadowed, for the State has recognised that the Medical Officer of Health cannot be omniscient. The Infectious Disease (Prevention) Act of 1890 (53 and 54 Vict., c. 34, sect. 4) says, 'In case the Medical Officer of Health is in possession of evidence that any person in the district is suffering from infectious disease attributable to milk supplied within the district, from any dairy situated within or without the district, or that the consumption of milk from such dairy is likely to cause infectious disease to any person residing in the district, such medical officer shall, if authorised on that behalf by an order of a justice having jurisdiction in the place where such dairy is situate, have power to inspect such dairy, and if accompanied by a veterinary inspector or some other properly qualified veterinary surgeon to inspect the animals therein, and if on such inspection the Medical Officer of Health shall be of opinion that infectious disease is caused from consumption of the milk supplied therefrom, he shall report thereon to the Local Authority, and his report shall be accompanied by any report furnished to him by the said veterinary inspector or veterinary surgeon, and the Local Authority may thereupon give notice to the dairyman to appear before them,' etc.

"Here there is provision for employing a class of veterinary surgeons apart from the veterinary inspectors, but if such surgeons are to perform their duties efficiently they must be properly trained, and they ought perhaps to be tested as to their special fitness for the work before they enter upon it. The veterinary colleges must therefore be ready to give additional teaching facilities, not only on the questions of meat inspection and the qualities of milk, but also in the geological and climatic conditions as they affect animals. More instruction must be given in hygiene, especially by a series of demonstrations as to good and bad hygienic conditions both in towns and in the country. A short course of lectures should also be given upon the principal laws and orders affecting the subjects which come under the care of veterinary hygiene. Such an extended course would present no difficulties here, for is not Professor Hobday our lecturer on the subject? After a proper course of training the Royal College of Veterinary Surgeons would no doubt soon see its way to grant a diploma of veterinary hygiene to those who were already members or fellows and who had duly passed a special examination.

"A specially qualified body of men being thus provided, places would have to be found for them throughout the country. At first, no doubt, this would be difficult on account of the expense, but the stockowners of the country form so large and important a body of men that as soon as they had satisfied themselves by actual proof that such a system saved them from loss they would readily assist in its extension. The districts over which the veterinary sanitary officers would have to preside should be large enough to occupy their whole time, and the remuneration should be sufficient to tempt the best men in the profession to accept the posts whilst they were still young. There should also be provision for a reasonable security of tenure. One of the great faults, both of the medical and of the veterinary profession, is that an individual, when he is once qualified and has obtained a moderate practice, no longer takes the trouble to keep himself posted in the advance of his profession. He therefore soon falls behind in his knowledge, and the more intelligent members of the community, finding themselves almost equally well informed upon some technical points, though they lack the groundwork, cease to look up to the individual, and so come to despise the whole profession. The best men in every profession are ever students, for they are always eager to acquire knowledge, and the more they know the more anxious are they to learn. For this reason only the very best men should be advised at first to enter the veterinary sanitary service. They will have to create the service. They will have to fight and to overcome many difficulties, both of fact and prejudice. Not only will the public at first be opposed to them, but as some of their duties must necessarily overlap those of the Medical Officers of Health, there is certain to be some jealousy between the less competent and more narrow-minded members of the two services. The more highly-educated and the more far-seeing members of the medical profession, knowing the disadvantages of dual control, will readily yield to the veterinary service those duties which seem especially to belong to it. They will welcome a release from comparatively alien subjects, as it will allow them more time for the effectual performance of their own work, which is already sufficiently arduous.

"The duties of the veterinary sanitary officers would be to carry out, in the first place, the provisions of the Diseases of Animals Act, and of the Dairies, Cowsheds, and Milkshops Orders of 1885 and 1886. These Acts and Orders, however, would be enlarged and extended, so that they would occupy the same relation to future veterinary legislation that Lord Morpeth's Health of Towns Act passed in 1848 now bears to the Public Health Acts. They would mark the awakening of the Government to the importance of legislation in matters concerning veterinary hygiene. Excellent work has already been done by veterinary surgeons in regard to the precautions which should be taken in the distribution and sale of the meat of diseased animals, or of milk from tuberculous cows. The question is one of international importance, but it has an economic as well as a hygienic side, and the two are in some measure antagonistic the one to the other. An attempt should be made to formulate an efficient system of meat inspection, though it is probable that such a system, based at first upon compromise, will only become effective after many mistakes have been made. Veterinary surgeons, by reason of their special training, are much more likely to distinguish the varying qualities of meat than are medical men. The veterinary sanitary officers therefore should be given full authority over piggeries, slaughter-houses, butchers' shops, and dairies, though it will often be necessary for them to consult the Medical Officer of Health before they decide what is or what is not fitted for human food.

"The veterinary sanitary service would also adopt a system of careful notification of all cases of infectious disease. If such a system were carefully carried out in connection with rabies, and if every owner of a dog was compelled to attach a registered label to it, to be renewed at first at short intervals, the ownerless dogs could soon be eliminated, and hydrophobia would become as rare as leprosy in England.

"The veterinary sanitary officers would necessarily require assistance, for they would control large districts. Such assistance would be provided by recruits from the class who now furnish the veterinary sub-inspectors. Reliable men who have held positions of trust in the district, and who are therefore well acquainted with the local inhabitants and their customs. Even these men, however, should receive some course of training before they enter upon their duties.

"The veterinary inspectors would remain, for they have done good and useful work. But, as vacancies occurred, their successors would be selected from the most energetic, tactful, and skilled of the veterinary sanitary officers, who would be tempted by the prospect of wider duties and better pay. The veterinary inspectors should form a more select body of men, specially qualified to sift evidence, and fitted by the highest scientific training to cope with the more formidable epizootics, which must from time to time appear, for they seem to be as inseparable from our present form of civilisation as such murrains have been from all past forms. The veterinary inspectors would be responsible directly to the department of agriculture, whilst the veterinary sanitary officers would be under the control of the County Councils. Both classes of officials should be wholly occupied in their duties, and should be prohibited from practising their profession privately, lest there should be even a suspicion that their interests as individuals might clash with the efficient performance of their public duties. The surest way to ruin the service would be to make a series of small appointments worth from £40 to £100 a year each. The occupants of these little posts would then be obliged to find other means of obtaining a livelihood, and they would make their veterinary sanitary duties subordinate to their other occupations. Whereas, if a service of well trained men devoted their whole time and all their energies to the study and practice of veterinary hygiene, we might hope to obtain in the future a great school of general or comparative pathology, useful alike to the State, to the community, and to the individual. Many preventible disease would be abolished, stockowners would become more prosperous, the average life of man would be further prolonged, and medicine would owe a still greater debt of gratitude to the veterinary profession" (applause).

"The Rt. Hon. Walter Long (Minister of Agriculture), who was loudly cheered on rising, said: Sir Howard Vincent, Mr Principal, and Gentlemen,—I have first of all to thank your Chairman for the kindly way in which he was good enough, in the first instance, to refer to my visit to your College to-day, and to thank him for his saying, as he was good enough to do, that by coming here I have given some evidence of the great interest which I take, both in my public and private character, in your great profession and in your great Institution, the Royal Veterinary College.

"Gentlemen, you will allow me to congratulate you upon the address which has just been delivered, and I believe I shall be permitted to ask you before I sit down, to vote your cordial thanks to the lecturer for his very interesting paper. I am sure it is almost unnecessary, except as a mere matter of form, that anybody should make such a demand at your hands. You have already conveyed to the lecturer, by the hearty applause with which you have greeted at intervals his address, and by the way you have welcomed him when he announced the conclusion of it, your appreciation of the wise and weighty words he has read to you, and you will, I hope, permit me to be the medium of conveying to him your thanks, and the thanks of everybody interested in the health of man, or of the brute creation, for the very interesting survey he has given us of the work which has been, and one in the past, and for the valuable suggestions he has made as to improvements that are possible in the future (applause). Gentlemen, I came here to listen, and not to speak, and therefore, my remarks will be very short indeed. I gathered at the commencement of these proceedings, that this meeting was very similar in its character to meetings that I have attended in days gone by, of other educational establishments. I remember that in those days when I was happy enough to be as young as many of those I see around me to-day, that there were several things we liked and a great many things we disliked; but of all the things that we disliked—and cordially disliked—at the meetings that then took place, there was nothing that we disliked so much as a long speech on any possible occasion (laughter). Though I daresay in some particular details there may be differences in these establishments, this is a common ground of agreement, in which all find themselves united, and, therefore, as I do not want on this first occasion of visiting your College to make a bad start, I have every reason for curtailing my remarks.

"Gentlemen, in the position which it is my privilege to occupy, my colleagues in office and myself are brought daily into very close connection with the great profession of which some of you are members, and to which some of you hope to belong some day, and no doubt, will adorn, and we have to reply enormously upon the assistance which we receive at the hands of the veterinary profession throughout the country. I need hardly say, for it is well known to you, that the Board of Agriculture has received in days gone by, and receives to-day, the most valuable assistance at the hands of Professor Brown, who is sitting near me, and who is so well known to us (cheers), which assistance to that department of the State it is impossible for any one to exaggerate, in regard to the readiness, generosity, and goodwill with which it has invariably been rendered. Mr Cope and Mr Duguid are two gentlemen whose services are of great value to us, and who bring us into, perhaps, still closer connection with your great calling. But it is in our administration that we are so dependent on the members of your profession, and I am glad to remember that during the time I have had the honour to be head of the department with which I am now associated, it has lain in my power to extend, at all events to some small degree, the opportunities of veterinary officers in connection with the work which they do for us throughout the country. We have, I hope, already given them opportunities, in connection with the administration of the Swine-Fever Acts, to exercise a wider discretion, which has certainly thrown upon them great responsibility, and I have no hesitation in saying that up to the present time I have had no cause, neither have my colleagues at the Board of Agriculture, to do otherwise than congratulate ourselves on the change which has been made, or the way in which it has been taken advantage of by the veterinary profession. I am glad, also, to remember that with regard to veterinary inspectors at ports we were already able last year to raise two or three gentlemen from temporary appointments to the permanent service, and no doubt we shall see our way, as time goes on, to extend and increase that number (applause). I only refer to those two facts as proofs, and I think I may fairly offer them to you as such, that the Board of Agriculture not only realises how dependent it is upon the veterinary profession for the successful administration of the Acts for which we are responsible, but also that we recognise fully the work that is done by veterinary surgeons throughout the country, and that we are most anxious, whenever we have an opportunity, to set our sail in regard to your work, by improving in any way that is possible the positions that veterinary officers hold, as showing the confidence we are able to feel in them (applause).

"I do not propose, as I have said, to make a speech, and I am certainly not going to endeavour to instruct you in the science of your profession, because I am afraid there would be several difficulties in the way if I were to make any such attempt. I am confident that I should scarcely have commenced before the students would be tumbling over each other in their anxiety to escape from the room (laughter). Therefore I will confine myself to one piece of advice, which I hope you will not think it an act of impertinence on my part to give you. It has been remarked by the Chairman, in his interesting and able opening remarks, that the veterinary profession has made great progress during recent years, and similar opinions were to be found in different portions of Mr D'Arcy Power's address. Well, gentlemen, it may be—I doubt not it is—a matter of wonder to you who are members of this great profession, that the community at large, and your fellow countrymen, have, apparently, been so slow to recognise the great advance which your profession has undoubtedly made. I have no doubt that when you dwell upon this fact—for a fact undoubtedly it is—you wonder at it, and at first, possibly, you are inclined to be dissatisfied and impatient; but if you will stop for a moment to realise what your fellow countrymen are, I think you will see that. after all, it is a very ordinary event. The British people, as you know, are very slow to move or to act, or to realise what are the changes that are going on around them. I am not a very old man, but I am old enough to remember those representatives of the profession who, in my young days, were called 'cow doctors'! (laughter). Some of them I remember were interesting and amusing men; but, I think, they would have been still more so had they been preserved for examination in some human museum (laughter). They were recognised authorities on the health and diseases of animals in their district; but to draw a comparison between them as they were, and the veterinary officers of to-day, I believe would tax the wit of even the most able orator that ever addressed a public audience. The change has been very remarkable. It is a change not merely in the scientific knowledge possessed, not merely in the power now possessed by veterinary officers of to-day, to lessen pain, to get rid of disease, and to detect the truth as to what is wrong, but it is a change in the whole character and position of the man—a change so great that I believe your countrymen have not yet recognised it, and they have not looked for that improvement in your position which, depend upon it, will not much longer be delayed (cheers). There is one word of advice which I would like, very respectfully, to give you as a layman. It is the opening remarks of your lecturer's address. It is to make up your minds, those of you who are going into the world, to take up your positions in various parts of it, that by your conduct in daily life, and the prosecution of your business, you will do everything to secure for yourselves personally that respect which your great profession has already won for you as a body (cheers). You are entitled to regard yourselves as scientific men who have struggled hard to equip yourselves for the profession you are going to follow; you are entitled to remember that you are endowed with the greatest blessing one man can impart to another, viz., the art of healing—of lessening pain, of checking and even defeating disease. I say, therefore, endeavour by your individual efforts to maintain that high standard in your public and private conduct that shall cause your profession, as a whole, to be recognised amongst the leading professions of this country.

"Let me congratulate you on this eve of your session. I wish it all possible success and prosperity, and to those amongst you who are not yet identified with any particular walk in life, I express the hope that you may have a most prosperous future. I congratulate you on having Professor M'Fadyean, whose name is in itself a power, as the head of this College; and, if I may, I will thank the Professor who has delivered his interesting address to us to-day. I have no doubt whatever that both the State and your fellow countrymen will recognise, either in legislation, or in daily life, the great position to which your distinguished profession is undoubtedly called (applause).

THE CHAIRMAN.—I am quite sure, Mr Long, that the Court of Governors, the Principal, and every one connected with the Royal Veterinary College must desire to thank you most heartily and warmly for the observations you have addressed to this gathering (cheers), observations which will be read with the greatest satisfaction and pleasure by every veterinary surgeon throughout the country, and which assuredly cannot fail to greatly advance the profession in the public estimation, which every well-wisher of it desires (applause). I am sure, also, it is not necessary to call upon any individual to second the motion made by the Minister of Agriculture, to give a hearty vote of thanks to Mr D'Arcy Power for the able and interesting address he has delivered. I am sure you will, one and all, second that motion, and show your appreciation of it by acclamation. Allow me in your names to return to Mr D'Arcy Power the thanks of all connected with the Royal Veterinary College for the valuuble and instructive address which he has delivered to us to-day (loud cheers).

MR D'ARCY POWER—All that I have to do is to thank you most heartily for the very kind words in which you have proposed the vote of thanks, and to you, gentlemen, for the way in which you have carried it.

THE CHAIRMAN.—I will now call upon Professor M'Fadyean to read the list of prizes that have been awarded during the past session.

PROFESSOR M'FADYEAN, the Principal of the College, having read the list of prizes (which appears elsewhere in this number), said:—"In addition to the list of competitions for which these College prizes are awarded, we are fortunate in having also an inter-collegiate competition. General Sir Frederick Fitzwygram, one of the Governors of this College, has, with great generosity, for a long period of years, offered annually three prizes of £50, £30, and £20 respectively. To the competition for these prizes the Principal of each of the veterinary colleges in this country is allowed to send two students, and I am very happy to be able to state that at the recent competition, two of the three prizes were secured by students of the London Veterinary College, Mr Harber having been awarded the first, and Mr Thurston the third prize.

In another respect the past session has been one of the most successful in the history of the College, for 70 per cent. of all the students who presented themselves for the professional examinations, passed. Taking into consideration the searching character of the examinations of the Royal College of Veterinary Surgeons, as now conducted, I think it will be admitted that such a large percentage of passes is eloquent testimony to the diligence of the students" (applause).

PROFESSOR BROWN C.B., in rising to propose a vote of thanks to the Chairman said:—"Gentlemen, in a very few words I want to call on you to perform a graceful act at the conclusion of these proceedings.

"Without a chairman a meeting of this kind would very soon resolve itself into a number of conjured elements, and we are also extremely glad that we have with us such distinguished members of the Board of Governors as the gentlemen who attend here to-day, who will leave their duties, however various they may be, and take the trouble of helping us in carrying on this work.

"For my own part I feel I am rather fortunately placed to-day in being allowed to propose a vote of thanks to the Chairman, because, speaking as an old attendant at these meetings, I cannot help feeling that this has been one of a special character (applause). We have for the first time in the history of the College the presence of the Minister of Agriculture (applause), and I doubt very much if any of us at the moment fully appreciate the great effect that his presence to-day is likely to have on the estimation in which the profession will be held throughout the country (hear, hear). Putting on one side the very kind remarks he has made, the mere fact of his coming here will help us forward in the right direction (hear, hear). The observations made by the Chairman and everything that has been said to-day, have been of a most interesting character, and your lecturer has given us hopes of the future to which the most ambitious mind could not do more that aspire. There is absolutely no limit to the hope he has given us in years to come. I cordially agree with him in nearly all he has suggested—at any rate in all the principles he has advocated, and I can only express the hope that all of us here may live to see the day when the two medical services may be so intimately connected that it will be a puzzle to distinguish the one from the other.

"Remembering the warning of Mr Walter Long about long speeches, and the possible results of them, I will not give one to-day, lest I should introduce a single discordant note into a number of the most harmonious compositions (laughter and applause). I will therefore call upon you to give, by acclamation, a vote of thanks to your Chairman" (loud cheers).

THE CHAIRMAN.—"It only remains for me most cordially to thank the learned Professor who has been so kind as to propose a vote of thanks, and you for the way in which you have recorded it. I have had an easy and very pleasant task in presiding at this meeting, and I hope, on behalf of His Royal Highness the Duke of Cambridge and the Court of Governors, that the session that is commencing now will be amongst the most prosperous and successful of all the sessions in the history of the Royal Veterinary College" (applause).

The proceedings then closed, and the company adjourned to another part of the building, where they were entertained at luncheon by the Principal and staff of the College.

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ON A SEPTICÆMIC BACILLUS FOUND IN THE OX.

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THIS bacillus was isolated from portions of the carcase of an ox which had been exposed for sale as human food at Smithfield Market, and subsequently seized. The meat was received from Dr Bond, Medical Officer of Health for Holborn, and the following note by his sanitary inspector describes the appearances of disease which led to the seizure of the carcase. "Four quarters of a young ox, weighing 50 stones, from Wavenden, Bucks, were found to be extremely soft, sodden, and wet, magenta coloured (varying from light to dark), and smelling strongly of drugs; there was much serous effusion and inflammation in the intermuscular and other tissues in many parts (muscles, glands, and kidney); the glands were enlarged and badly congested; the left kidney was inflamed, full of blood, and a large cyst was embedded in it; the right kidney had been removed. The meat was ill-bled, and the animal was evidently killed when moribund." The carcase, it should be added, was perfectly fresh.

Immediately the meat was seized Dr Bond sent the following portions to the British Institute of Preventive Medicine for bacteriological examination. (1) A kidney which showed a number of small hæmorrhages into its substance. There was a simple retention cyst in the cortical portion of the gland, which had obviously nothing to do with the fatal illness. (2) A lymphatic gland with some surrounding fat; the former was distinctly congested, and there were one or two small hæmorrhages into its substance. (3) A small portion of the diaphragm, which also showed a few minute hæmorrhages into the muscular substance. (4) A portion of a skeletal muscle sodden and dark-coloured.

In a stained cover-glass preparation of the juice expressed from the kidney a few bacilli were to be seen. Gelatin plates were inoculated with muscle and kidney juice, and from the resulting cultures a coccus and a motile bacillus were isolated. At the same time two guinea-pigs were inoculated under the skin with an emulsion made by shaking up small portions from the interior of the muscle with sterilised normal saline solution. Both these guinea-pigs died on the fifth day with double pneumonia, and a bacillus identical with that isolated from the plate cultures was obtained in pure culture from their heart-blood. The bacillus isolated from the plate cultures was similarly tested on guinea-pigs, and gave exactly corresponding results. In stained cover-glass preparations made from the pleural juice of each of these animals a bacillus could be seen which morphologically and in its staining properties resembled the bacillus seen in the cover-glass preparation made from the original kidney. The coccus isolated from the gelatin plates proved to be non-pathogenic for guinea-pigs, and its presence was assumed to be due to an accidental *post-mortem* contamination of the meat.

MORPHOLOGY.

The micro-organism when seen in the blood or tissues of an inoculated animal appears as a shortish bacillus with rounded ends. Under these circumstances it is of fairly constant length, and individuals may exhibit a tendency to bi-polar staining. When grown on artificial media, on the other hand, a marked degree of pleomorphism prevails, as a reference to Plate III., Fig. 1 will show. The predominant type is a bacillus measuring from $1{\cdot}25\,\mu$ to $1{\cdot}5\,\mu$ in length with rounded ends. Shorter rod shapes, almost oval bacilli, and occasional coccal forms also occur. Two bacilli may rarely be seen joined end to end, but chains of greater length are never seen. Spore formation was not observed ; the bacillus is actively motile.

STAINING PROPERTIES.

The bacillus stains well with the ordinary basic anilin dyes. Methyl-violet and watery carbol-fuchsin particularly gave very good results. The bacillus is rapidly decolourised by alcohol after staining by Gram's method. Although culture experiments show that it is always present in large numbers in the heart-blood of inoculated animals, it is extremely difficult to demonstrate its presence there by staining methods. With well-prepared blood films it is only now and again that one may see a single or perhaps two bacilli in the microscope field. In the pleural, peritoneal, and tissue juices of inoculated animals, which have been killed before becoming actually moribund, the bacillus can be readily demonstrated.

CULTURAL CHARACTERISTICS.

The following are the principal points noted in connection with the culture of the bacillus on various media :—

Gelatin plate cultures, at 22° C. ; the colonies are visible to the naked eye at the end of the second day, and attain their maximum size by the end of the third or four day. They appear as small raised white spots with sharp-cut edge, and are quite round, measuring on the third day about ·25 mm. in diameter. Viewed under a low power and by transmitted light, they are seen to be finely granular in substance and distinctly brownish in colour. Colonies in the depth of the gelatin are of a uniform deep brown colour and rather opaque; surface colonies show a deeper brown central portion and a lighter brown peripheral zone. The edge of the colony is quite even and well defined (Fig. 2).

Gelatin streak, at 22° C.; very faint indication of growth at the end of twenty-four hours; at the end of forty-eight hours the growth appears as a narrow raised streak with well-defined edges which show no tendency to spread. The growth attains its maximum between forty-eight and seventy-two hours. Viewed by direct illumination the streak has an opaque white appearance, viewed by transmitted light it appears of a brownish colour. There is no liquefaction of the medium.

Gelatin stab at 22° C.; at the end of forty-eight hours there is a fine white growth along the entire length of the needle track with a small "pin-head" expansion at the surface end.

Gelatin shake, at 22° C.; on the second day a faint wavy cloudiness is seen in the medium. By the fourth day small round colonies may be seen in the depth of the gelatin, whilst the same are sprinkled over the surface.

Agar streak, at 37° C.; free, moist-looking, opaque, greyish-white looking growth with spreading edges at the end of sixteen hours. The growth increases in extent up to the end of about forty-eight hours, by which time the condensation water at the foot of the agar slope is opaque with growth. On agar at 22° C. growth is much slower than at 37° C., but considerably more active than on gelatin at the same temperature.

Glycerine-agar streak; growth is perhaps a little more active than on plain agar at corresponding temperatures.

Alkaline litmus-agar streak, at 37° C.; distinct colour change at end of twenty-four hours, deep red colouration at end of forty-eight. No subsequent discharge of colour. At 22° C. no colour change is obvious until the end of at least forty hours.

Alkaline litmus-sugar agar streak, at 37° C.; reddening much more pronounced at the end of twenty-four hours than in the case of plain litmus-agar. After seventy-two hours a gradual discharge of colour occurs, commencing at the bottom of the tube. This reducing action extends about half-way up the agar, and above that level the red colouration is reduced in intensity, but not discharged.

Sugar-agar stab, at 37° C.; active gas development by twentieth to twenty-fourth hour. At 22° C. gas development occurs some hours later.

Potato, at 37° C.; towards the end of the second day an almost invisible moist-looking patch may be distinguished on the surface; the area of the patch does not seem to increase in extent, and after several days assumes a faint yellowish-brown tinge. Growth on potato is feeble and uncertain, being not always obtainable.

Alkaline litmus-milk, at 37° C.; distinct colour change at end of twenty-four hours. The red colouration then gradually decreases and all colour is discharged by the fifth or sixth day. At 22° C. the colour change does not occur in the first instance until several hours later than at 37° C.

Peptone beef broth, at 37° C.; at the end of sixteen hours there is a general cloudiness of the broth. By the end of the second day there is a small stringy deposit at the bottom of the tube. By the sixth or seventh day a considerable number of bacilli have lost their motility, and fallen to the bottom in small clumps. By the tenth or eleventh day there is a thick sediment of clumped bacilli, and the upper part of the fluid is appreciably clearer. By the twentieth day the upper part of the fluid is to the naked eye quite clear. On examining a hanging drop preparation of the clear fluid a few bacilli may still be seen in it. One or two of these may show a little sluggish movement, but the majority are absolutely motionless. A few days later the clear upper part of the fluid will be quite free from bacteria. The growth does not at any time form a pellicle on the surface, nor does it show any tendency to adhere to the sides of the tube. The reaction of the broth, originally alkaline, becomes strongly acid within twenty-four hours of inoculation. The acidity, as roughly tested by the reaction with blue litmus paper, begins to decrease on the third or fourth day, and by the fifth or sixth day the broth has again become feebly alkaline. The cultures were tested for indol up to the twenty-first day with negative results.

Figures 3 and 4 in Plate III. show subcultures from infected animals. Figure 3 shows the free growth obtained on agar at 37° C., and Figure 4 the much scantier growth on gelatine at 22° C. The tubes were inoculated by passing a platinum wire lightly over the surface of the medium, and were formalised at the end of twenty-four and seventy-two hours respectively.

EXPERIMENTAL INFECTION OF ANIMALS.

The pathogenic action of this bacillus was tested on tame mice, white rats, guinea-pigs, rabbits, and young bull calves. For the purpose of inoculation either an actively virulent subculture (between two and six days old) from the heart-blood of a previously inoculated animal, or a bacillary emulsion, made by shaking up a forty-eight hours' old culture on agar with 5 cc. of sterilised normal saline solution, was used.

Tame mice died between fourteen and eighteen hours after the subcutaneous injection of ·1 cc. of the culture. Death was the result of a septicæmia, and the bacilli were recovered from the heart-blood and pleural juice. The only change noted *post-mortem* was some excess of fluid in the peritoneal cavity.

White rats were less susceptible to the action of the bacillus than any of the other animals used. They were obviously very ill twenty-four hours after the subcutaneous injection of ·3 cc. of a culture, but death did not result from this dose and the animals were usually quite well again in three or four days.

In the case of medium-sized guinea-pigs also a dose of ·3 cc. was used, and was invariably followed by a fatal result. When the injection was made into the muscles of the thigh death occurred within forty-eight hours, and usually towards the end of that period. The only changes to be seen *post-mortem* were a bloody œdema at the site of inoculation, and injection of the peritoneal membrane, with some excess of fluid in its cavity. When a similar dose was injected into the subcutaneous tissue death occurred on the fourth or fifth day with double pneumonia. The animals became comatose towards the end, and often died in convulsions. Subcultures from various organs and from the blood showed that the bacillus was universally distributed. A doe guinea-pig which was inoculated proved to be pregnant, and in that case the bacillus was found in the amniotic fluid. Microscopically the condition of the lungs varied from one in which a number of small hæmorrhages were scattered throughout their substance to that in which large patches of pneumonia involved one or two lobes. Other changes usually present were capillary injection of the peritoneal and pleural membranes, with excess of fluid in the cavity of the former. Less commonly small hæmorrhages into the kidney substance were seen. Microscopic examination of sections of the lungs showed that the pneumonia was primarily a hæmorrhagic one.

With large rabbits a dose of ·5 cc. was invariably followed by death within twenty-four hours, the greater number of those inoculated dying within eighteen. The animals died comatose, and death was preceded by a marked fall in body temperature. The bacillus was found universally distributed in the blood, and the only tissue change found after death was an extensive bloody œdema at the site of inoculation. In the case of rabbits it seemed to make no difference in the fatal result whether the injection was an intra-muscular or a purely subcutaneous one.

Three bull calves were inoculated with the bacillus. One survived the operation and ultimately recovered after a long period of ill health, one died a few hours after inoculation with a general infection of the blood, the third died on the eighth day with a well-marked septic pneumonia. The following notes give the more important details with regard to these several calves.

Experimental Calf No. I., about six weeks old. This calf was inoculated with 2 cc. of a broth culture (twenty-four hours old) from the heart-blood of a rabbit. The injection was made on 26th January 1897 into the subcutaneous tissue on the left side of the neck. On 27th January the animal seemed poorly, there was some swelling about the site of inoculation, temperature in rectum 39·8° C. On 28th January there was no change in the general condition, but there was now at the site of inoculation a definite flattened swelling with a circular outline measuring about 8 cm. in diameter; temperature 39·8° C. On 29th January the swelling was slightly more prominent; the animal had diarrhœa and was voiding a little blood-stained mucus; temperature 40·5° C. On 30th January there was still free scouring; the swelling was very hard and presented no sensation of fluctuation; temperature 40·2° C. During these days the animal had been taking its food fairly well, but had lost a great deal of flesh. Nothing special occurred until 15th February when an attack of synovitis developed in the left fore knee. In the middle of April the calf was still in very poor condition; the knee joint was still distended with fluid, and the character of the swelling on the neck remained unaltered. Shortly after this the animal's condition improved, and the synovitis passed away leaving an apparently good joint. On 14th May, the calf being in very good condition, I decided to explore the swelling on the neck, which presented itself as a hard, flattened, circumscribed tumour with circular outline and freely movable between the skin above and the neck muscles beneath. On cutting into it I found a hard, white, oblong mass of necrotic tissue measuring about 4 cm. by 2·5 cm., and about ·75 cm. in thickness. This was lying quite loosely in a smooth-walled cavity which was free from pus or fluid of any kind. The operation wound healed up very quickly, and all trace of swelling disappeared. The calf was kept under observation until the end of July, and appeared to have made a perfect recovery.

The necrotic mass when removed was quite free from any smell, and contained no living bacteria. Microscopically it consisted chiefly of altered striated muscle fibre derived from the panniculous carnosus and a certain amount of connective tissue. Here and there faintly stained degenerated forms of bacilli could be seen. The specimen was interesting from a pathological point of view as being a perfect example of necrosis without suppuration, a condition which in the human subject, at anyrate, is quite uncommon, but is met with occasionally in the "quiet necrosis" of bone.

Experimental Calf No. II., aged about three weeks. In the case of this animal 2 cc. of a broth culture (forty-eight hours old) from the heart-blood of a rabbit were injected into the external jugular vein at 11.15 A.M. on 15th February. At 3.30 P.M. the calf was found to be moribund; it was bringing up a little blood-stained fluid, apparently from the lungs. It died at 4.30 P.M., just five and a quarter hours after the injection. The body was opened at six o'clock the same evening. There was no sign of thrombosis in the vein into which the injection had been made. Both lungs were extremely congested and œdematous. The right lung showed a small irregularly shaped hæmorrhage at its internal border; this had not the appearance of being a hæmorrhagic infarct, and it may be added, in view of the rapidity with which death occurred, that there was no sign of pulmonary embolism. There was a little blood-stained fluid in the right pleural cavity. There was also a recent hæmorrhage into the ligament representing the remains of the umbilical vein. A subculture on agar taken from the blood an hour and a half after death gave a very free growth of the bacillus (Fig. 3).

Experimental Calf No. III. This was a particularly fine looking pedigree animal between four and six weeks old. It was inoculated on 12th April with ·75 cc. of an emulsion made by shaking up a two-days-old culture on agar from the heart-blood of a rabbit with 5 cc. of sterilised normal saline solution, the injection being made into the external jugular vein. The animal seemed fairly well up to 17th April. During the six days it took its food well, its respiration was rather hurried, but there was no abnormal chest sounds. There was no swelling or induration at the site of inoculation. The temperature was elevated, but unfortunately the record for these days was mislaid. On 18th April the animal was obviously very ill, lying down and refusing to stir. Its respiration was very hurried, its temperature was 40·5° C., and it was still taking its food. On the morning of 19th April it seemed a little better, but its temperature was 41·2° C. At mid-day it was moribund, and it died in the evening after its temperature had risen to 41·8° C. The body was opened next day. A double pneumonia was found, the greater part of both lungs being in a condition of red hepatisation. Here and there were distinct small hæmorrhages into the lung substance. The pneumonic process was especially marked at all the borders, and throughout the upper lobes. There were several small yellowish-white necrotic patches along the borders. A central part in each lower lobe was not pneumonic, but merely congested and œdematous. The bronchial glands were swollen, and appeared to be in a condition of almost purulent infiltration. There was a slight excess of peritoneal fluid. The kidneys were rather paler than is normal; scattered throughout their substance and especially numerous in the pyramidal portion were a number of greyish nodules rather larger than a pin's head. Cultures of the bacillus were obtained from the heart-blood, the pleural juice, and the kidney. The microscopic examination of sections of the lungs showed that the general condition was one of fibrinous pneumonia. Sections through a necrotic area showed patches of altered tissue which stained a faint yellowish-green colour with methylene blue. In the necrotic patches isolated bacilli which had taken the stain deeply were seen lying about. Sections through the grey nodules in the kidney showed the same appearance of necrotic tissue and isolated bacilli. Had the animal survived a little longer these nodules would doubtless have assumed the form of multiple miliary abscesses. There was no thickening about, or thrombosis in, the vein into which the injection was made.

The results of these inoculation experiments and the conclusions to be drawn from them may be summarised as follows:—

(1) White rats were not very susceptible; they suffered a temporary illness and recovered.

(2) Rabbits and tame mice with a subcutaneous, and guinea-pigs with an intra-muscular, injection, died within forty-eight hours. The cause of death was an acute simple septicæmia unaccompanied by any gross tissue change; in other words the decomposition of their own proteid constituents and the consequent evolution of toxic substances was so rapid that the animals were poisoned before reaction effects in the tissues had time to show themselves.

(3) Guinea-pigs, after an injection into the subcutaneous tissue, died on the fourth or fifth day with a double septic pneumonia added to the general septicæmia. In these cases the bacilli entered the general circulation more slowly, the guinea-pigs were rather less "susceptible" than the rabbits, the septic process was more deliberate, and sufficient time elapsed before death occurred for the local effects of the poisonous decomposition products to become manifest.

(4) With regard to the calves—Calf No. I., survived a subcutaneous inoculation. I think in this case it is clear that the bulk of the bacilli injected into the subcutaneous tissue never travelled beyond the site of inoculation, but were checked by the intensity of the local reaction —to speak in general terms. The actual dose of bacilli which entered the general circulation was so small that the natural powers of resistance were in the result equal to the stress thrown upon them, and the animal recovered after a prolonged illness, including an inflammation of a synovial membrane and necrosis of the area of tissue into which the bacilli were injected. Calf No. II., had a very large number of bacilli thrown directly into the circulation; the bacilli, as the culture experiments show, are capable of extremely rapid increase at the body temperature, and the result was a septicæmia even more acute than

in the cases of the rabbits and mice. Calf No. III., which appeared to be a stronger animal than either of the others, received a medium dose directly into the circulation. But sufficient time elapsed before death occurred, not merely for a local inflammatory reaction in the lungs to manifest itself, as in the case of the guinea-pigs which survived inoculation for five days, but for a further stage to be reached—necrosis of the poisoned tissues.

In carrying out the inoculation experiments it was found that there was a considerable diminution in the virulence of broth cultures after the seventh day; a diminution which, it may at once be said, was probably due to the rapid dying out of the bacilli rather than to the lowering of the pathogenic activity of individual bacilli. Thus a beef-broth culture ·5 cc. of which on the sixth day killed a large rabbit in sixteen hours appeared to be quite inert on the tenth day in a dose of 2 cc. This result was tested several times, and no variation occurred. Such a culture of ten days' growth, however, proved fatal to a rabbit after two injections of 2 cc., each with an interval of sixteen hours. The animal died within twenty-four hours of the second injection, and a subculture from its blood gave a characteristic growth which possessed the normal virulence. After passing the original culture through a considerable number of guinea-pigs and rabbits, it was found at the end of eleven months to possess just the same degree of virulence for these animals and for mice as it did at the commencement.

All attempts to infect mice, rats, and guinea-pigs by the mouth failed.

SOME BIO-CHEMICAL REACTIONS.

Some experiments were carried out to determine the decomposition products resulting from the action of this bacillus upon carbo-hydrates and proteids.

For the former the medium was prepared as follows:—

Glucose	40 grammes
Peptone	10 ,,
Sodium chloride	7·5 ,,
Calcium carbonate	20 ,,
Distilled water	1000 ,,

The flask containing this was steamed on two successive days for forty-five minutes. After inoculation the flask was incubated for twenty-one days at 37° C. The following results were arrived at. A large proportion of the glucose had undergone decomposition, only about 4·5 grammes remaining unaltered in solution. From the contents of the flask succinic and lactic acids were recovered, but were not estimated quantitatively. About 1 cc. of alcohol was recovered by distillation; this gave the iodoform test and was probably ethyl alcohol. The volatile fatty acids recovered were small in amount, about ·1 gramme expressed in terms of acetic acid; they were not separated qualitatively. There was no evolution of sulphuretted hydrogen.

In order to study the proteid decomposition the following medium was prepared. Five hundred grammes of lean beef were finely minced and added to 1000 cc. of a ·75 per cent. sodium chloride solution in a flask. The flask was steamed as before. Its sterility having been tested, the flask was inoculated and kept at 37° C. for twenty-eight days, during which time active growth of the bacilli took place. The results

Fig. 1

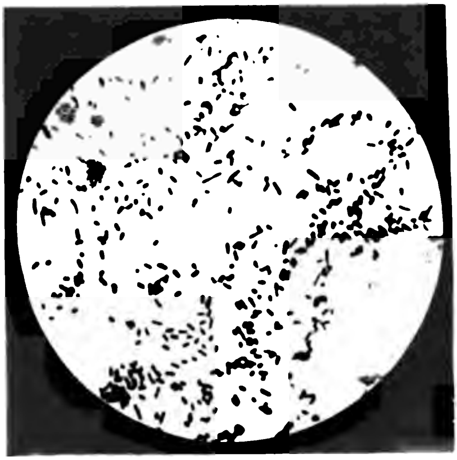


Fig. 2

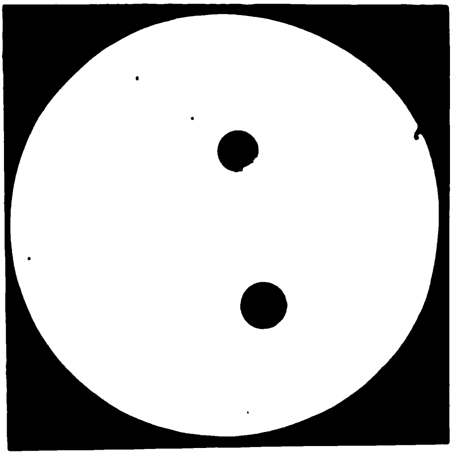


Fig. 3

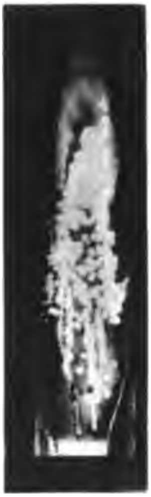


Fig. 4

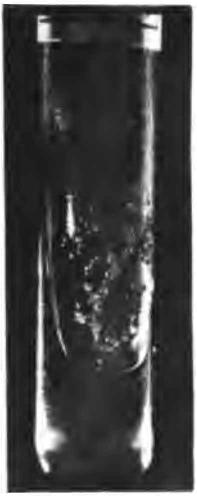
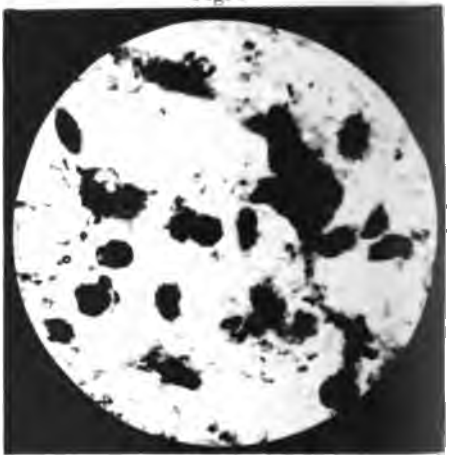
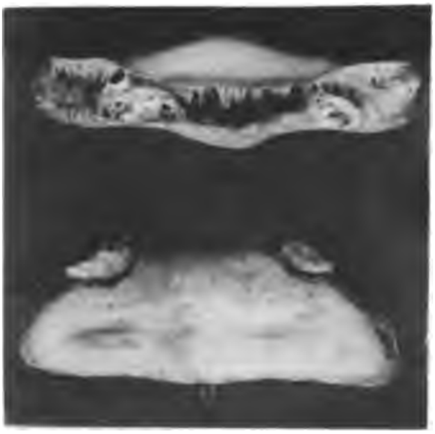


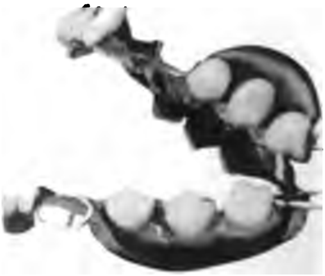
Fig. 5



(For description of Figs. 1 to 5, see opposite page.)



DISEASED NAVICULAR BONES.
VET. CAPT. PEASE'S CASE.



ARTIFICIAL TEETH FOR DOG.
PROFESSOR HOBDAY'S CASE.

of the examination of the contents of the flask were mainly negative. There was no production of indol, phenol, or skatol, nor were mercaptans or sulphuretted hydrogen present. There appeared, however, to have been some production of ammonia during the growth of the culture. The exact nature of the acid produced in proteid media, as noticed in reference to beef-broth cultures, was not ascertained owing to the dying out of my cultures during a temporary absence from the laboratory, and before my researches in this subject were completed. This production of acidity from proteid matter is of some interest when considered in relation with the rapid pathogenic action of this bacillus in the rabbit, an animal which is known to be extremely sensitive to any alteration of the reaction of its blood in that direction. That the bacillus has no peptonising action is indicated by the fact that no liquefaction of gelatine occurs.

In conclusion I have only to add that this bacillus does not belong to the so-called "hæmorrhagic septicæmia" group of bacteria, to which it may possibly bear some superficial resemblance. Nor can I ascertain that it corresponds with any other pathogenic bacillus hitherto described. It has, then, been thought worth while to record its characteristics rather minutely in the hope that it may be identified again.

My very best thanks are due to Professor M'Fadyean of the Royal Veterinary College for the facilities afforded me at that Institution for carrying out the calf inoculations which have been referred to herein.

DESCRIPTION OF PLATE.

FIG. 1. Cover-glass preparation from culture on agar × 1000.

FIG. 2. Colonies from gelatin plate on fourth day × 20.

FIG. 3. Smear culture on agar from blood of calf, incubated for twenty-four hours at 37° C. (Natural size.)

FIG. 4. Smear culture on gelatin from pleural juice, incubated for seventy-two hours at 22° C. (Natural size.)

FIG. 5. Section through necrotic patch in lung of calf dying on eighth day after inoculation, stained with mythylene blue, × 1000.

Photographs by Mr Bernard from Mr Foulerton's preparations.

(For description of the other two figures see pages 370 and 362.)

THE PROPHYLAXIS OF GLANDERS OF THE HORSE.[1]

By Professor NOCARD, Alfort Veterinary College.

THEME.

1. Every horse which presents any symptom whatever indicating the existence of glanders or farcy ought to be submitted to the mallein test. If the mallein provokes a complete reaction (both organic and thermal) the horse ought to be declared glandered, and slaughtered without delay; if, on the contrary, the mallein does not provoke any reaction, the horse ought to be declared not glandered, whatever may

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[1] **A communication made to the Thirteenth International Congress of Medicine, held at Moscow, from the 19th to the 26th September 1897.**

be the appearance of the symptoms or of the lesions which it presents.

2. When a horse is recognised glandered all the horses which have cohabited with it ought to be submitted to the mallein test. After the test they ought to be divided into two groups, as follows.

The first group will include the healthy animals, that is to say, those which have not given any reaction, organic or thermal. The owner will be allowed the free use of these; they will be kept in a special stable which has been thoroughly disinfected, and into that stable one will not introduce new animals without having them submitted to the mallein test.

The second group will comprise all the suspected horses, that is to say, all those that have reacted to mallein in a more or less complete manner. These horses will be marked and a record kept of them; they will be rigorously isolated from healthy animals, and kept in a stable which has been thoroughly disinfected. They will have special attendants, with grooming utensils, halters, drinking troughs, pails, mangers, and nosebags, kept exclusively for their use. Every month or every two months they will be submitted to a new injection of mallein; those which, in addition to a reaction to mallein, develop any clinical sign whatever of glanders or of farcy will be slaughtered without delay. On the other hand, those which shall have undergone two successive injections of mallein without reacting will be declared healthy, and placed at the free disposal of their owner.

This procedure is actually followed in France. It has the great advantage of reducing as far as possible the sacrifices imposed on the owner; it enables him to keep a notable proportion of the contaminated animals, whose lungs at the time of the first test contained specific lesions, recent or disseminated. In fact, an experience which is already extensive proves that many of these animals, when withdrawn by isolation from every opportunity of new contamination, rapidly recover from the pulmonary lesions with which they were attacked. It would therefore be excessive to require their slaughter, so long as no external symptoms arises to show that the disease is progressing and on the point of overcoming the resistance of the animal.

Besides the measures applicable to stables that are known to be infected, there are some others of a more general nature, which would permit the discovery of suspected animals, and diminish the chances of infection of healthy animals. These are as follows:—

1. In the countries where military commissions make a periodic census of the horses fit for service in the army, these commissions would render great services in pointing out to the sanitary agents those horses which present symptoms indicative of glanders.

2. An effective supervision of fairs and markets, of horse slaughter-houses, and of knacker-yards, would permit the recognition of a good many unknown centres of contagion, and the application to them of sanitary measures capable of promptly stamping them out.

3. Horses kept for hire, and those of commercial travellers, hawkers, boatmen, travelling showmen, etc., being more particularly exposed to the risk of contracting glanders, ought to be the object of a periodic sanitary visit. They ought not to be allowed to circulate without a certificate of health, delivered by the Sanitary Authority. This certi-

ficate ought to be valid for only a definite period, which should be as short as possible; whenever required it ought to be presented to the Sanitary Authority or the Mayor of the Commune.

4. Finally, stables attached to inns ought to be thoroughly disinfected as frequently as possible, and notably on the day following fairs and markets held in the locality. The disinfection ought to be directed specially to the troughs, mangers, hayracks, flooring, rings used for tying up, pails, corn chests, and nosebags, in common use.

DEVELOPMENT.

The preceding theme contains an outline of the principles which in my opinion ought to govern the prophylaxis of glanders of the horse, and I shall now endeavour to briefly justify them.

I. The whole system is based on the methodical employment of mallein. In effect, mallein provokes in horses that are affected with glanders in any degree a series of general and local phenomena which are designated by the name of the reaction, and when mallein provokes in a horse that is suspected a reaction that is at once thermal and organic one may affirm that the horse is glandered. When, on the contrary, mallein does not provoke any reaction it may be affirmed that the animal is not glandered whatever may be the appearance of the lesions which it presents. Mallein has thus enabled me to differentiate from glanders and farcy several affections which formerly were confounded with them.

With regard to this first point there is no longer any room for argument; thousands of observations and experiments establish the incomparable value of mallein in diagnosis.

II. Glanders very rarely attacks only one animal in a stable which contains a certain number of horses; the rule is that other animals adjacent to the first one become in their turn glandered, but they may for a long time retain all the appearances of health, and when the clinical signs of the disease become apparent they have for a long time been disseminating around them the germs of glanders, and have thus in their turn contaminated other horses previously healthy.

Mallein enables one to ascertain in a few hours which animals of an infected stable are really contaminated, and which on the contrary have escaped infection; hence it follows that one is able to separate the healthy animals from the diseased and to remove them from all risk of contagion.

In a stable where the disease has been recognised only after a considerable lapse of time the number of horses that react to mallein is sometimes considerable; that is because, contrary to what one might believe in view of the extreme delicacy (fragilité) of the glanders bacillus, the disease is *mainly* propagated by way of the alimentary canal. It suffices to cause a healthy horse to ingest together with his solid or liquid aliment a small quantity of virulent glanders culture in order to render it glandered in a few days. Before the experiment the animal does not react to mallein; eight to ten days afterwards it reacts in the most decided manner, and if it is then killed one finds in the lung miliary tubercles in every way similar to those of the natural disease. It is this which constitutes the whole danger of common drinking troughs, which are the rule in the army and in the majority of large stables. A glandered horse has only to snort there for a good many healthy horses which drink after him to be in turn affected. Herein lies the great danger of inn stables, where horses from all parts succeed one another without even the precaution being taken to clean out the troughs, mangers, or hayracks.

Certainly glanders may arise by all methods of inoculation, as, for example, by the deposition of a glandered discharge on a wound or excoriation, by the friction of an infected sponge even on an intact mucous membrane, or by the employment of a surgical instrument contaminated with glanders pus. One can experimentally communicate glanders to a healthy horse by subepidermic inoculation, by subcutaneous, intravenous, or intratracheal injection, by pulverisation into the trachea, etc.; but if at the *post-mortem* examination of a horse infected in one of these ways one finds the lungs filled with glanders lesions, these lesions are not miliary tubercles similar to those of the natural disease, but more or less extensive infarcts, or multiple and circumscribed areas of glanderous pneumonia or broncho-pneumonia, more or less analogous to those which one encounters in the ass after inoculation with a glanderous product.

I repeat that the only means of experimentally obtaining miliary tubercles in the lung identical with those of natural glanders is to cause the animal to ingest virulent matter, culture, discharge, or pus.

I have experimented on thirty-two horses, one ass, and two mules, and I have always obtained the same results. The whole thirty-five animals have taken glanders from the third to the eighth day after ingestion of the virulent material. In seventeen of them the existence of glanders during life was revealed only by the reaction to mallein; in eighteen others the disease was manifested by the ordinary clinical signs.

III. When mallein has pointed out which animals in a suspected stable actually have internal glanders lesions, although presenting no external symptom, it might seem that one ought not to hesitate to have all these animals slaughtered, However, I recommend that only those that present some clinical signs should be slaughtered. With regard to the others, whatever their number may be, one may keep them carefully isolated from healthy animals, and use them as before; it is only necessary to submit them every month or every two months to the mallein test, and when they have supported two successive tests without reacting one may return them to the ranks and put them at the free disposal of their owners, for they will then have completely and definitively recovered from the glanders lesions which they carried in their lungs.

What! can a glandered horse recover?

Certainly it may recover, and the occurrence is far from being rare.

When I first spoke of the curability of glanders I raised a veritable outcry. I had dared to touch the *dogma of the incurability of glanders of the horse.* In order to overcome the violent and almost universal opposition provoked by my proposition I had to multiply experiments and accumulate facts.

Be it well observed that I have never pretended that all glandered horses may recover; on the contrary, I think that glanders, as it was formerly known, ought to be considered as practically incurable. When the submaxillary glands are enlarged, and nasal discharge or nasal or cutaneous ulcerations have appeared—which was what one formerly called the beginning of the disease, but which was in reality the "beginning of the end"—one may say that the animal organism has been overcome in the struggle which it has waged against the invading microbe; the cells have been powerless to prevent the generalisation of the glanders bacillus, and there is so little chance of their regaining the upper hand that it is better to have the horse which is clinically glandered slaughtered at once as incurable and dangerous. But glanders with external symptoms—the only stage of the disease with which we were formerly acquainted—is only the last phase of the disease; the first is represented by these nodular lesions of the lung, the extreme frequency of which in infected studs has been pointed out to us by mallein. It is these initial lesions, discrete and limited, which have been provoked by the accidental and unrenewed ingestion of food materials soiled with discharge or pus. These, I say, are the lesions which may evolve silently in the depth of the lung, and which very frequently end in final retrogression and a definite cicatrisation without one having at any moment suspected their existence. The facts which prove this are very numerous. One of the most demonstrative and the one best known, because it has provoked numerous and violent discussions, is that which concerns the Remount Depôt of Montoire.

Glanders appeared there in the first months of 1892, and the Minister of War ordered all the horses of the depôt to be submitted to mallein. A Commission was charged with the carrying out of the operation, and with the making of a report on the results obtained.

Besides the horses which presented external symptoms indicative of glanders, mallein provoked a reaction in a considerable number which presented absolutely no signs permitting one to consider them suspected. On various occasions the Commission had slaughtered a certain number of these horses. On the 21st of August 1892 it also caused the slaughter of eleven belonging to a group of those which had not reacted to mallein. In all the animals killed, whether they had reacted to mallein or not, there were found identical pulmonary lesions, consisting mainly in greyish or transparent miliary tubercles, without caseous centres, without fibrous capsules, and without an inflammatory ring at the periphery.

The glanderous nature of these tubercles was admitted by all the members of the Commission save one. That is what explains one of the conclusions of the report, namely, "The horses which have not reacted to mallein ought not to be considered as free from glanders;" but what the report does not state is that, while entirely affirming the glanderous origin of these tubercles, I then expressed the opinion that if these horses did not react to mallein, that was because they had recovered from the glanderous lesions with which they had been attacked, that is to say, the tubercles found in their lungs were no longer virulent, and no longer contained living glanders bacilli. This opinion, which was characterised as heretical, has been confirmed by all the facts observed since then.

The eleven subjects in question had been taken at random from a group of 105 horses in the Montoire Depôt which had not reacted to mallein. One was therefore entitled to believe that the ninety-four horses of the same group left alive had also in their lungs lesions of the same nature; in other words, all the Montoire animals must at a given moment have been more or less gravely infected.

When one remembers that this was a depôt where the horses are being constantly changed, where the promiscuity is absolute, where the animals are entirely free, day and night, both in the stable and at grass, to smell each other, to bite, to eat out of the same rack and drink from the same trough; and, above all, when one reflects that glanders had existed here undetected for nearly five months, one understands well enough that at a given moment all the horses in the depôt must have had glanders tubercles in the lungs.

Nevertheless, when all the horses, without exception, were first submitted to the mallein test (28th-31st May 1892) a certain number of them did not react, namely 97 out of 233. That was because for several weeks all the horses had been picketed in the meadow of the depôt; the abandonment of the infected locality, the rigorous individual isolation, the suppression of every opportunity of new contamination, the permanent life in the open air, and good food, had already permitted a certain number of the subjects to recover from the minute and sparsely distributed lesions of which they were the subjects. The proof of this is that on the occasion of the subsequent injections the number of animals which did not react to mallein went on increasing each time, until, finally, seventy-eight of the Montoire horses which mallein had declared glandered or suspected could, after having been picketed for five or six months, be redistributed among several different regiments and put to active service without any one of them developing the slightest disquieting symptom.

In short, what happened in the case of the Montoire horses is what always takes place in the horses of any corps or troop gravely infected with glanders. After several months of rigorous individual isolation applied to the whole of the animals, the horses are sent back to service without, as a rule, glanders reappearing. The only difference to be noted is that in the case of Montoire one knew that all, or almost all, the horses had pulmonary lesions at the moment when they were picketed, whereas up to that time no one had ever made, or been able to make, the same observation.

Analogous facts have been observed in Russia. In 1893, 658 horses of a brigade of the reserve cavalry of the government of Charkoff were submitted to the mallein test. In the case of only 290 of them was the test entirely negative; nevertheless, after several months of observation and isolation, and after the slaughter of a small number of suspects which had become clinically glandered, the majority of the survivors, having ceased to react, were sent back to service, and not one of them afterwards presented the least symptom of glanders.

The following is a fact of the same order, but still more convincing, which I obtained in 1894 in the depôt of a tramway company of Paris. Several cases of glanders having been observed, all the horses of the depôt, to the number of about 160, were submitted to the mallein test; of twenty-nine which reacted only three presented insignificant symptoms, and when these were slaughtered the autopsy revealed in them old glanders lesions. Of twenty-six others which did not present the least symptom twelve of the oldest and least valuable were successively slaughtered; in all of them there existed pulmonary tubercles, variable in number, and many of them translucent. One was therefore entitled to affirm that the fourteen other horses, equally healthy in appearance, but which had like the others reacted to mallein, had also pulmonary lesions. Nevertheless these were kept isolated in a special stable and injected twice a month with mallein. After the first month eleven of these horses ceased to react, and since then mallein has no effect on them.

They have been sent back into the midst of healthy horses, and they have done very hard work without any one of them ever having presented the least symptom of glanders. When submitted again to the mallein test after ten months of work not one of them has reacted; one may therefore consider them as completely and definitively recovered.[1]

But clinical observation never gives more than a certain sum of probabilities approaching more or less closely to certainty. In a subject of this nature one cannot accumulate too many proofs.

To the facts which I have just cited, I am in a position to add some new ones, which I hope will leave no doubt in your minds.

Last year the Military Commission of Hygiene and Veterinary Medicine of our country obtained from the Minister of War authority to make experiments tending to verify the exactitude of the facts which I have just exposed to you.

A first series of experiments had for object to establish:—(1) The reality of infection with glanders by the digestive passages; (2) The diagnostic value of mallein; (3) The possibility of provoking in the lungs of healthy horses the development of miliary tubercles in all stages of their evolution, and notably translucent tubercles, the glanderous nature of which has for a long time been contested.

This set of experiments was carried out on a series of six horses belonging to a regiment in which glanders had not been observed for ten years. None of these horses had reacted to mallein; two of them were set apart as control subjects by the Commission charged to carry out the experiments. The other four received the product of a potato culture of very virulent glanders bacilli mixed with half a pail of water. Fifty hours after the ingestion the temperature of these four horses rose suddenly from 1·8° to 2·6°; an injection of mallein practised on the sixth day provoked in the four subjects an organic reaction so intense that for three days it was feared that they would die. On the contrary, the control subjects supported the injection of mallein without any reaction. From the eighth day the four experimental animals had the submaxillary glands swollen, and one of them presented some nasal discharge and ulceration of the septum nasi. On the fifteenth day after the ingestion the Commission had two of the glandered subjects and one of the control animals slaughtered.

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[1] Of three others two in succession were killed and found to be glandered on *post-mortem* examination. The third is still living; for nearly three years it has borne the mallein injections without any reaction. It also has ultimately recovered from the serious lesions with which it was attacked.

The autopsy was made with the greatest care; the two glandered subjects had the lungs crammed with a large number of miliary tubercles in all stages of development, *and many of them entirely translucent.* On the contrary, the lungs of the control subject did not contain any tubercles, translucent or of any other nature.

The three other horses were killed at the end of three months. The control animal had no lesion in the lung or elsewhere. The two others, which for a long time had presented all the clinical signs of chronic glanders (swelling of the submaxillary glands, nasal discharge, nasal ulcerations), had their lungs literally crammed with tubercles in all stages of evolution; along with an incredible number of calcareous, fibrous, and caseous tubercles, many were found of a soft consistence and translucent appearance, without central softening, without condensation of the tissue at their periphery, and, in a word, identical with those which are of recent formation. By a veritable auto-infection incessantly renewed these horses had every day added to the number of their pulmonary lesions by soiling their liquid or solid aliment with their own discharges.

A second series of experiments was carried out on twelve horses, chosen like the preceding from a regiment which had for a long time been free from glanders. When submitted on two occasions to the mallein test none of these twelve horses gave any reaction. On the 30th November 1896, in the presence of the Commission charged with the execution of the experiments, each of these twelve horses was made to ingest an equal quantity of virulent glanders culture.

Three potato cultures, four days old, were carefully diluted in 300 cubic centimetres of sterilised water; each of the twelve horses drank in presence of the Commission half-a-pailful of water into which one had thrown 25 cubic centimetres of the glanderous dilution.

Fifteen days afterwards all these horses were again submitted to the mallein test. All reacted in the clearest manner.

The horses were kept under careful observation, and every month, or about that, the mallein test was repeated. Several of these horses presented clinical signs of glanders, and had in succession to be slaughtered, namely, three on the 21st January 1897, and two on the 16th May following. These five horses had never ceased to react to mallein, and at the autopsy the whole five presented glanders lesions, formidable in one of them, rather discrete in two others, and considerable in the two last.

From the end of February the nine surviving horses were picketed in the park of the Alfort Veterinary College; they have since remained there, day and night, in all weathers, save for the four or five days necessary for each mallein test.

On the 5th June a sixth horse had to be killed on account of an attack of very severe double acute pleurisy. This horse had not given any reaction on the occasion of the last injection of mallein, and it was then considered as recovered.

At the *post-mortem* examination a small number of miliary tubercles, fibrous, caseous, or calcareous, scattered through the superficial strata of both lungs, were discovered. These were carefully collected, and to them was added a small focus of recent broncho-pneumonia of undetermined nature; then, after having pounded the whole, one half of the pulp thus obtained was injected under the skin of the neck of a donkey, and the other half into the peritoneum of two male guinea-pigs. Two other guinea-pigs received each 5 cubic centimetres of the pleuritic liquid. All these animals, which were inoculated nearly three months ago, are to-day quite well. It is therefore certain that the specific lesions from which they were inoculated were no longer virulent.

The six remaining horses had not for a long time reacted to mallein, and they also had to be considered as recovered.

On the 11th July last the Military Commission had four of them killed, and the autopsy was made in the presence of a large number of military and civil veterinary surgeons from Paris and the neighbourhood. In the four subjects one observed the existence of indisputable glanders lesions, although these were rather sparing in number; the majority of the tubercles were fibrous, some caseous, others semi-translucent. All the pulmonary lesions which could be found were carefully collected from each horse; they were pounded as completely as possible, and one half of the pulp thus obtained was injected under the skin of the neck of a healthy donkey, and the other half into the peritoneum of two male guinea-pigs.

I left Alfort on the 14th August last, and on that day the four donkeys and the eight guinea-pigs inoculated on the 11th July were still quite healthy, and they had never been diseased. It is therefore certain that the glanders tubercles from which they were inoculated no longer enclosed a single living glanders bacillus.

Experimentation has confirmed the indications of the mallein test, and justifies one in affirming that these four glandered horses have completely recovered.

There remain two horses which for a long time have not reacted to mallein. We ought to believe that if they were slaughtered they would be found to be in the same state as the others.

They have been kept in order that they may be sent back to service; they are on the point of going through the extensive manœuvres of the North. If they come victoriously through this test, as I firmly believe they will, it will no longer be possible to pretend that the recovery of these horses is only apparent, and that their lesions, for the moment dormant, might be re-awakened under the influence of heavy work and repeated fatigue. The experimental demonstration will thus be more complete and striking.[1]

The measures which I recommend have not only the support of experimentation; they are also sanctioned by long practice.

In France they have been regularly applied in the army since the 20th September 1895, and the experience acquired demonstrates their efficacy.

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[1] These two horses, yoked to heavy waggons, have borne admirably the manœuvres of the North, which persistent rain rendered very trying. On their return they were lively, their coats were shining, and they had not lost a kilogramme of their weight; at no moment had they presented the least sign of malaise or fatigue. On their return to the Veterinary College they were again submitted to the mallein test, and neither the one nor the other gave an organic or thermal reaction. The Commission had them killed on the 24th October; the autopsy showed in their lungs some miliary tubercles, numerous in one of them and rather rare in the other, for the most part fibrous, some infiltrated with lime salts, and some others grey and slightly translucent. As in previous cases, these lesions were collected and carefully pounded, and the pulp thus obtained from each was injected under the skin of the neck of an ass, and into the peritoneum of two male guinea-pigs. No doubt these experimental animals will comport themselves like the others, which to-day, three months and a half after inoculation, are in a flourishing state of health.

There is as yet no law imposing them in every case, but horse owners very soon grasp their importance, and there are very few of these who refuse to allow them to be applied in their stables. The results obtained are excellent.

I could multiply examples in proof of this, but I will confine myself to the following. In the month of July 1895, the Compagnie Generale des Voitures of Paris requested me to organise measures against glanders, which decimated its horse stock. The situation was grave; out of a total of 12,000 horses distributed in twenty-three depôts, 586 showing symptoms of glanders had to be killed in six months (July 1895 to January 1896); in the month of August alone 170 were killed.

I obtained from the directors, thanks to their eminent President, permission to submit the whole of their horses to the mallein test. This was no light undertaking, for it was necessary to operate without interfering with the work of the Company. In each depôt it was necessary to separate the healthy animals from those which were contaminated, as shown by the mallein test; and it was necessary to distribute them in special stables with attendants and utensils for their exclusive use. Above all, it was necessary to interdict their access to common drinking troughs, which are, unfortunately, the rule in France in all the great studs of horses; finally, it was necessary to disinfect all the stables, and in the case of those with wooden mangers and partitions it was necessary to substitute cement for the wood, so as to render disinfection possible.

The Company granted me permission to do all that I considered necessary. The operation lasted eight months; it was carried out in the best conditions by three of my pupils, MM. Schrader, Guignard, and Malet, under the admirable direction of one of my old fellow students, M. Blanc, head of the department.

Out of 10,231 horses submitted to the test, 2037, or in round numbers, 20 per cent., gave the characteristic reaction. This percentage though so high does not quite show the gravity of the disease, for out of twenty-three depôts belonging to the Company sixteen were almost exempt from the disease; in the seven others the number of contaminated horses varied from 32 to 56 per cent.

Out of these, 2037 animals which were denounced by the mallein test although none of them presented the least symptom indicative of glanders, 687 became clinically glandered and had to be slaughtered (440 between July and December 1895, 152 in 1896, and 15 in 1897); on the other hand, 338 *have ceased to react to mallein, and have been returned to the category of the healthy.* A large number of these have had to be cast on account of old age, the wear and tear of work, or accidents. We have made a *post-mortem* examination in each case; in the whole of them the lesions were manifestly cured, being fibrous or calcareous. In a certain number of animals these lesions were carefully collected and used to inoculate culture media or animals; the cultures have not given Löffler's bacillus, and none of the inoculated animals have taken glanders.

The operation would not have been complete if we had not submitted to the mallein test the horses purchased to recruit the stock. Out of 5027 horses submitted to the committee for purchase in 1895 mallein denounced 237, or 4·71 per cent., and these were returned to the dealers.

In 1896 the proportion of suspects reached 6·77 per cent. of the inoculated horses, that is to say, 381 out of 5626. Do not these figures eloquently demonstrate the extreme importance of this measure?

Mallein has not only the advantage of enabling us to make an early diagnosis of pulmonary glanders, which is inaccessible to any other means of diagnosis, and of thus rendering us masters of the disease; it has also permitted us to return to work a large number of horses which one would formerly have had to isolate as suspects on account of symptoms indicative of glanders. From the 11th October 1895 to the 30th June 1897, 627 horses had thus to be submitted to the mallein test (392 on account of enlargement of the submaxillary glands, 63 on account of nasal discharge, 156 on account of lesions simulating the swellings, lymphangitis, or farcy buds, 2 on account of epistaxis, 14 for various lesions). Out of these 627 horses only 265 have been recognised glandered and slaughtered; 362 were not glandered, and could be immediately returned to work.

The following figures will give a good idea of the results obtained by the systematic application of these measures. They refer solely to the horses presenting external symptoms of glanders. The number of animals killed in this way in the last half of 1895 was 586; they fell gradually to 159 in the first six months of 1896, and to 140 in the following six months, and only 62 were killed in the first half of 1897.

It ought not to be forgotten that out of these 62 horses killed in 1897, 15 were already glandered without symptoms in 1895; the figure ascribable to new infection is therefore only 47? That is a rather low figure, normal one might say in view of the conditions of work of the Company's horses. While waiting at the public stands for cabs they are constantly in contact with all sorts of horses; contrary to the orders given to their drivers they often drink from the common pails kept at the stands or at public houses. They are thus incessantly exposed to infection, but the measures which are taken warrant the statement that such accidental infections, which will take place from time to time as long as all the stables of the cab proprietors have not a clean bill of health, will remain limited, and provoke only small unimportant outbreaks, easily stamped out, and that the serious state of affairs existing in 1895 will not occur again.

Thus it is seen that the application of the measures which I recommend has in practice yielded the results that theory, based on experiment, permitted one to anticipate.

TETANUS.

By SIDNEY VILLAR, F.R.C.V.S., Harrow.[1]

IN introducing to your notice the subject of tetanus, I feel an apology is due from me for asking you to consider so common a disease, one with whose clinical symptoms we are all so familiar—too familiar, probably, in the case of many of us—but I am prompted to do this by the frequent allusions now being made both in the medical and lay press to the new method of treatment for the prevention and cure of this terribly painful and frequently fatal disease; and I hope that to some extent my paper, and in a great degree the discussion which may follow, will serve to bring to our minds some detail which individually we may have overlooked or forgotten, so that at our parting we may be conversant with the most recent theories as to the actual cause of tetanus, the manner in which the pathological conditions are produced, and the steps which may be taken to prevent altogether, or remedy as much as possible, such conditions.

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[1] Read before the Royal Counties Veterinary Medical Association, November 1897.

Unfortunately, the animal with which in veterinary practice we mostly have to deal—the horse—is very susceptible to tetanus. The ox does not take it readily; in a somewhat large cattle practice I have only seen three cases in that class of animal, two of which supervened on the operation of castration, and the third on a cut fetlock. Sheep also appear to be resistant, although I remember an outbreak in which a number of lambs were affected with the disease, the primary wound being the unhealed umbilicus or navel.

The dog appears rather insusceptible. A clever theory to account for this was advanced by a friend of mine recently. It was to the effect that a dog always cleanses his wounds with his tongue, and so removes the tetanic poison. This is, however, probably not the correct explanation, for it is found that when a dog or cat is inoculated experimentally with the tetanus organism it is difficult to get an animal affected, so that both dogs and cats appear to be naturally resistant to this disease.

I have never seen tetanus in pigs. A case is recorded in the *Veterinarian*, vol. xlvii., where a pig recently castrated died of lockjaw, and upon a *post-mortem* examination being made some ashes were found in the scrotal wounds. Two cases in pigs are recorded in the *Veterinary Journal* of 1890, and five cases occurring simultaneously on one farm after castration are given in the *Veterinary Record* of 10th July last. We must, however, remember that young pigs are very frequently affected with epilepsy, the symptoms of which to some extent resemble those of tetanus.

I have already used the terms *tetanus organism* and *tetanus poison*, so that perhaps this is a fitting opportunity to refer to these in detail.

In 1884 Nicolaier found that when a small particle of soil from the streets or the fields was placed under the skin of mice, rabbits, or guinea-pigs, symptoms of tetanus of a progressive character followed in a few days, and that after death a small abscess was found at the point of inoculation. In the pus of this abscess was an organism which, when re-introduced into a similar small animal, again produced tetanus, and it has since been demonstrated that if the pus from the primary wound of a person affected with tetanus be in a similar way inoculated into mice, rabbits, or guinea-pigs, tetanus supervenes, and exactly the same bacillus as that got from inoculation with soil is found to be present.

This organism, which is known as the tetanus bacillus, has since been absolutely separated from all other organisms found in the pus of wounds; it has been artificially cultivated apart from all such organisms, such cultivations when inoculated into animals have produced tetanus, and upon *post-morteming* the inoculated animals the organism has again been separated from the pus of their wounds.

It has been laid down as an axiom and is accepted generally by bacteriologists that a bacillus which successfully passes through such a cycle as this establishes its right to be recognised as the specific organism, the rightful cause of its particular disease, and I think it can be accepted without doubt that a bacillus is the specific cause of lockjaw; we may go further and say that no case of tetanus can naturally arise without this bacillus being present in the animal body.

The bacillus is named from its shape the "drum-stick bacillus," for it consists of a little filament or thread with a dilated rounded end, this rounded end being the reproductive particle or spore. The organism is of course very minute. Its other distinguishing characters are that it is motile and anærobic, that is, it cannot live in an atmosphere containing oxygen; its spore develops best at the temperature of the blood. When cultivated artificially it liquefies gelatine, and has a characteristic mouldy sort of odour. In what is known as a stab culture, made by pushing a soiled platinum needle into a test tube of gelatine, there is a peculiar method of growth, branching in a particular way from the needle stab, and resembling the branches of a spruce-fir tree. By its shape and such characteristics as these it is easily recognised from other bacilli.

When the tetanus bacillus gains entrance to the body through an abrasion of the skin or a contused wound, it is found that under favourable circumstances it multiplies, and it is then to be found in the pus of the wound and in the infiltrated tissues immediately around the wound, but it never exists in large numbers. In this it is unlike the bacillus of anthrax, for only a few of the organisms are ever present; they are invariably confined to a localised area, and are never to be discovered in the blood or tissues, in the internal organs, or central nervous system.

This limited localisation of the bacillus is accounted for by the fact, as we have seen, that it will not live where oxygen is present, and it therefore only multiplies in the lower layers of pus of a wound, where it is cut off from the oxygen in the air and also from that contained in the blood of the capillary blood-vessels.

As with other bacilli, the spore or reproductive particle will remain alive long after the other portion of the organism has perished (as long as twelve months), and only awaits a favourable opportunity to grow and develop. These spores of the tetanus bacillus seem to have a remarkably wide distribution; Bassano found them in twenty-seven out of forty-three samples of soil gathered in various parts of the globe. This observer made experiments which led him to assert that soils which contain much organic matter almost invariably contain tetanus bacilli, and that defective drainage and imperfect general hygienic conditions are favourable to its existence.

Mr Stanley, the explorer, mentions in his work the fact that the native inhabitants of Central Africa dip the points of their arrows in the soil of certain swamps, with the positive result that their wounded enemy will die of lockjaw. The same method of poisoning their arrows is resorted to by the aborigines of the New Hebrides and other islands in the Pacific Ocean, and, recognising this, the French Government at the present day inoculate their soldiers to prevent them falling victims to this remarkable sagacity of the savage tribes.

But although the spores of the tetanus bacillus are widely distributed in the soil, some districts appear to be free from them; hence the fact that tetanus is not met with in all localities.

Thirteen years ago, that is long before the pathology of the disease was understood, Mr Joseph Woodger, in a paper read before the Central Veterinary Medical Society, pointed out the remarkable prevalence of tetanus amongst horses used in dust carts; out of ninety-six cases which he recorded thirty-seven were in horses used in dust carting, and in eight consecutive years he found that in this class of horse the number affected amounted to 3 per cent. per annum, whereas in omnibus horses stabled in the same neighbourhood only $\frac{1}{14}$th per cent. per annum became affected. Mr Woodger was convinced that the fact of so preponderating a number of cases being found amongst dust contractors' horses was to a great extent due to poisonous emanations from the decaying heaps of refuse always present in dust contractors' yards, and one cannot but admire that gentleman's foresight and correctness of judgment, in view of the fact that at the time he wrote the tetanus organism had not been recognised.

In my own practice in Middlesex there are two farms where the disease is specially prevalent. One of these, at Alfreton, is occupied by a farmer who habitually spreads on his fields large quantities of London refuse; at the second farm, 5 miles away, the bailiff bought two large loads of the sweepings from London roads in 1893, and dressed his home meadows with it. On these meadows his colts and young stock were pastured, and for two and a half years tetanus was endemic on his farm; during this period six colts became affected, and I believe only one entirely escaped the disease.

It will doubtless occur to you that animals used or pastured where such refuse is deposited would be more than usually likely to be wounded by broken glass, nails, etc., and therefore more liable to tetanus, but it is an interesting fact that out of Mr Woodger's thirty-seven cases in only four horses were any wounds discoverable.

But the disease may be of an endemic character on farms and places altogether innocent of refuse heaps and dust contractors, and not only on particular farms and premises, but in particular stables. I knew an instance where a veterinary surgeon attended in one stable three horses affected with the disease at the same time.

In a note to the *Veterinarian* of 1894, Mr Wheatley of Reading, in recording a case of tetanus, says, "this horse was lying on peat moss, which probably conveyed the contagion," and he adds, "since horses have been stabled on this litter I have had more cases of tetanus from wounds than I have ever had before." Theoretically, this is exactly what we should expect, and it will be interesting to have the practical experience of others as to this; for myself, I have had cases occur in stables where moss litter is used, and also where straw forms the bedding, but I always suggest that a wounded horse should not lie on peat moss, the more especially that in my neighbourhood there is somehow an idea amongst horsekeepers that peat moss is liable to cause lockjaw.

Dr White, a gentleman practising in Ceylon, where from the heat of the climate the natives wear next to no clothing, and lie about a good deal on the ground, tells me that in that country almost every wound is followed by tetanus. In veterinary practice the ordinary operation for umbilical hernia is well known to be frequently followed by tetanus; this used to be ascribed to the irritation of the ligature used, but I think we must now attribute it to the wound being in contact with the soil when the animal lies down. As in most country practices, docking horses is a rather frequent operation with me, and it is noteworthy that in the only case in which tetanus has supervened the animal on its way home after the operation broke away from the man in charge, and got embedded in the morass adjoining the Welsh Harp Reservoir at Hendon. It was with some difficulty got out of the mud, and was noticed to be affected with tetanus on the tenth day afterwards; it ultimately died.

Death in this disease appears to be actually due to one of three causes.

Firstly, tonic contraction of the respiratory muscles.

Secondly, to broncho-pneumonia associated with fœtid breath, and arising from the flow of putrid saliva, which cannot escape from the mouth, down the trachea into the bronchi.

And thirdly, from starvation and exhaustion.

The next point which arises is, how do a few infinitesimally small bodies, such as this bacillus, in a wound, or in a well-defined and localised area round a wound, produce such direful effects. The answer to this is, and it has been experimentally proved to demonstration, that the bacillus during its existence and multiplication in a wound elaborates chemical poisons of a most deadly character, and that these poisons are absorbed into the system and produce the widespread effects. The process is therefore an intoxication or blood poisoning, and tetanus is now recognised as an "infective" disease. It must be remembered that the bacillus is a vegetable organism, indeed is a plant, and then it will be more clear how in the course of its life history it can elaborate poisonous alkaloids, just in the same way as the nux vomica tree produces strychnia, and the cinchona bush quinine. These and many other poisonous substances are, we know, built up by vegetable cells, and exert a specific action on animals. Just as strychnia exerts its injurious effects through the nervous system, so do the poisons formed by the tetanus bacillus act.

It is an interesting fact that when an experimental animal is inoculated with a quite young artificial growth of the tetanus organism the disease does not at once supervene, but if the inoculation is made with a growth, say ten days old, when some of the bacilli have had time to mature and perform the natural functions of their existence, tetanus rapidly follows. During the performance of such natural functions the active poison is, as we have already seen, produced.

It is probably due to the fact of the tetanus organisms forming their poisons so slowly that so many wounded animals, in what may be considered infected localities or stables, escape the disease, for practically we know that the organism must be introduced into the system of many wounded animals, and produce no ill effects. The explanation appears to be that the poison is produced so slowly that in healthy tissues, such as in a clean cut or punctured wound, the tissue cells destroy the organisms introduced long before they have time to form sufficient poison to produce the nervous symptoms of tetanus; whereas in cases of contused wounds and punctures, such as are produced by pieces of wood, etc., the tissue is so injured that the phagocytes or tissue cells are weakened, and are no longer strong enough to contend with the invading organism. The latter then multiplies and develops its poisons, which are absorbed and give rise to those reflex spasms and convulsions which we recognise as tetanus.

For the existence of tetanus it is necessary that a wound of some sort, either of the skin or mucous membrane, must have occurred. Tetanus is always traumatic, and the term idiopathic, which denotes arising without obvious cause, cannot properly be applied to this disease. The actual cause is the drum-stick bacillus, but the wound by which it gained entrance to the system may not be apparent; indeed the primary wound may be in the mouth, œsophagus, or any part of the intestinal canal. A case is reported by Ceicholi, in which a costive horse whose fæces were covered with blood-tinged mucus, became affected by the bacilli gaining entrance through the abraded surface of the rectum. It is probably that the presence of worms, sand, and foreign matters in the intestines, which were formerly assigned as causes of tetanus, produce wounds of the mucous membrane, and so allow the bacilli to gain an entrance.

The experiments of Solmani, who fed very susceptible animals with large quantities of pure culture of the bacilli, proved that the virus of tetanus when introduced into the unwounded digestive canal produces no ill effects, and he also demonstrated that the gastric and intestinal juices do not destroy the virus, for he produced tetanus from the fæces of an animal which had been fed on the virus.

Cases are also recorded where tetanus has followed wounds of the mucous membrane of the genital tract produced in the process of parturition or abortion.

Recognising that the bacillus is the cause of tetanus, we cannot now admit that the disease can be produced by exposing an animal to cold, or by over-exertion or fright, nor from neglect in the treatment of, or the application of irritating medicaments to, wounds.

For the purpose of considering its treatment, tetanus may be said to be either of an acute or subacute form, the acute including those cases in which distressing symptoms of great severity rapidly occur after the animal is first observed to be ailing, and which usually end in death about the fifth or sixth day, whereas in the subacute form the symptoms gradually develop and the spasms and convulsions are not severe. Such cases do not usually reach their greatest severity until about the ninth day, and in my experience the majority of them recover; in these cases the ninth or tenth day seems a critical period, as it is not rare to find fatal lung complications develop about that time.

Tetanus appears to supervene on trivial wounds quite as readily as after severe injuries. Wounds of the feet, limbs, and head have in my practice been more often the origin of tetanus than those on the body. There is no definite period of incubation in this disease. The earliest period that I can call to mind at which tetanus has supervened on an injury is the sixth day, and the latest period just over three weeks. The interval which elapses between the reception of an injury and the occurrence of manifest symptoms does not influence the course of the disease; whether the period be short or long it is equally liable to take an acute or subacute course. Cases are recorded where tetanus has supervened an hour or so after the infliction of an injury, but bearing in mind the *modus operandi* of the specific organism one must think that a primary wound was previous in such cases, and the second injury a coincidence.

Very interesting reports have recently been published where tetanic horses have recovered after being treated by injection with antitetanic serum, the recoveries being presumably due to the beneficial action of that agent. I have used it as a curative in two cases only, and in both the horses have died; in each the disease was of an acute character, and had manifestly been in existence for at least three days before injections were made. The first case, a hunter, was injured on 26th December, and it was observed to be stiff and unwell on the 7th January. On the 10th 100 cc., that is about 3½ ounces, of serum from an immunised horse were injected into the muscles of the neck, and he was placed in slings. At this time the symptoms were very marked; the horse was sweating and the paroxysms were frequent and severe, the superficial muscles being "as hard as a board." Twenty-four hours afterwards, at which time he received another 100 cc. into the opposite side of his neck, there was a very distinct improvement; the horse's tail was flaccid and could easily be moved in any direction, the ears were less stiff, the feel of the body was much softer, the spasm of the masticatory muscles was somewhat less tonic, and there was unquestionably more capability of movement of the jaws. I was delighted with the prospect, but all hope was lost on the third day, for pneumonia and a fœtid breath were then very evident. Somewhat foolishly, perhaps, a third injection of 100 cc. was made then, and a fourth of 30 cc. on the fourth day, but the horse died during that night. The second case was that of an aged pony to which attention was first asked on the 5th of the month, when all the usual symptoms of acute tetanus were shown. This animal received injections of 2½ F. drachms of antitetanic serum on the 6th, 7th, and 8th, but showed no remission of symptoms throughout, and died on the 8th of the month. There was no obvious wound in this case. The serum was obtained from a firm of chemists.

During the present year, in addition to the above, I have had five horses with tetanus. Two of them being of little value were at once destroyed; the other three have recovered and are now at work. All three were subacute cases; two of them were slung and given a little belladonna, also some nitrate of potash when their legs became swollen from standing. The third case, a well-bred mare about ten years old, was at grass throughout the attack, and recovered without treatment. Recently cases of spontaneous recovery in the horse have been recorded, and I have had previously two cases similarly recover without treatment.

In the three favourably ending cases above referred to the serum treatment was not employed, and it would be unfair to judge of its utility from its having failed to cure in two very severe cases, especially as there was delay in having recourse to the treatment. Nevertheless, the published experience of persons who have used it on a very extensive scale can but lead us to the opinion that, although it is capable of causing an abatement of the severity of the symptoms, it cannot be looked upon as a specific cure for tetanus when once that disease has developed.

In a recent number of *The Journal of Comparative Pathology and Therapeutics* (Sept. 1897), it is stated that out of nineteen cases of tetanus in the horse treated at the Berlin Veterinary College by intravenous injection of tenanus antitoxin, between June and September of this year, sixteen cases have proved fatal. In the same publication is a most interesting and instructive communication by Professor Nocard, who in a series of experiments injected large doses of the antitetanin into the jugulars of five horses which were only just beginning to show symptoms of tetanus, yet all five horses died of the disease. It will be remembered that it was this gentleman who first brought antitetanin to the notice of the veterinary profession; for over two years he has been an enthusiastic worker with the serum, yet so recently as August last he wrote that "the cure for tetanus has still to be discovered," and that "he who by injecting serum counts on curing the majority of his patients will expose himself to a cruel disappointment." He further observes that in the practical use of serum by veterinary surgeons the percentage of recoveries has not been higher with its use, but that in cases where it has been injected the convulsions have been less numerous and less intense, and when recovery has supervened convalescence has been more rapid.

In the *Veterinary Record* of 2nd October last, Major Nunn records the case of a nine-years'-old mare very severely affected with tetanus. She received eight doses of serum at intervals of twenty-four hours, and made a very rapid recovery. Other successful cases in which recovery has been rapid are recorded by Messrs Fraser, Reeks, Heatley, Sparrow, and others.

The usual duration of an attack in the horse is six weeks, and there appears no doubt that this period is considerably shortened by the systematic use of antitetanin, which acts as an antidote to the poison that is being continually formed by the bacilli at the wound. Having regard also to this constant formation of toxin, it is advisable if possible to eradicate the focus of the disease. In cases where the disease follows on docking, this is easily done by amputating a further portion of the tail, but in all other positions it is difficult except by making use of caustics, and these in our patients must be unsatisfactory. In addition to subcutaneous doses of antitoxin, I strongly advocate putting the animal in slings, and ensuring him as perfect quiet as possible, a laxative and nutritious diet, and as medicine only an occasional diuretic. I am altogether an unbeliever in the use of aloes, as it nauseates and takes the horse off his feed at a period when it is absolutely necessary that he should take all the nutriment the state of his jaws will allow.

A Continental authority advises the injection of the serum directly into the jugular vein, and claims that by so doing there is a gain of twenty-four hours in the treatment of the disease. I have never seen or heard of this being done in this country, but bearing in mind the want of success in the Berlin and Paris cases already quoted, where this method of procedure was employed, I am sceptical as to the statement.

One detail should perhaps be mentioned, namely, that where it is necessary to inject large quantities of serum, say 50 or 100 cc., it is advisable to plunge the needle of the syringe right into some of the muscles, preferable into those of the neck, for if such quantities are placed quite subcutaneously there is a danger of the skin sloughing.

Although one is not now in a position to ascribe great curative power to antitetanic serum, there is very strong ground for recognising it as a very certain preventative of the disease. I have in numerous instances injected wounded horses, both with the consent and without the knowledge of the owner, and have never seen any ill effect from its use. On one of the two farms to which allusion was made in the earlier portions of this paper, and on which usually about twenty-three horses and seven colts are kept, there occurred in the five and a half years previous to 1st July 1896 ten cases of tetanus, namely eight cases in horses, one in a bullock, and one in a donkey. This disease has existed on the farm for the last twenty years, and is in fact the bugbear of the place. I cannot call to mind a single case of external injury during the period I have attended prior to 1st July 1896 that was not followed by tetanus. Since that date there have been three cases of wounds in horses on this farm which I have treated; the first in a gelding, a severe laceration of the skin and flexor tendons just above the fetlock of the near fore leg, then a mare with badly broken knees, and thirdly a two-year-old colt with a deeply cut wound in his heel caused by barbed wire in a fence. Each of these animals has been injected, two of them twice, and one on a single occasion with antitetanic serum. Each has continued in perfect health and shown no sign of tetanus. Of course I cannot pretend to say that they would otherwise have been victims to that disease, but having regard to the history of their surroundings there is a very great probability that some of the three would have been attacked.

Professor Nocard says that when employed as a preventative antitetanic serum is absolutely efficacious, and one cannot read his article and the statistical tables which he gives without being struck by the fact that he has the very best of grounds for coming to this conclusion.

Tetanus is very prevalent in some localities in France, and the Professor has distributed antitetanic serum to sixty-three veterinary surgeons, who have treated for the prevention of tetanus 2727 animals with it. Of the animals so treated only one became affected, but during the same period the sixty-three veterinary surgeons saw 259 cases of tetanus in animals not so injected. Moreover, it is noteworthy that the horse which did become affected was not injected until five days after being pricked in shoeing, and although the delay was too great to prevent the appearance of tetanus the disease was quite of a mild type; so slight indeed was the attack that the horse was able to resume work within a fortnight.

As a preventative from 2½ drachms to 5 drachms of the serum should be used; it should be injected as early as possible after the wound is inflicted, and the dose should be repeated after an interval of ten or twelve days.

I can give no details as to the period of immunity which is conferred by the serum. One would, however, expect it to be of only short duration seeing that the active immunity conferred by a natural attack of the disease is only of a transient nature. Our worthy President, Mr Wheatley, has recorded the case of a horse which recovered from tetanus, but at an interval considerably under two years was again attacked with that disease. Personally, I treated a black filly which recovered from an attack in September, but again fell a victim to the disease in the following July; this case occurred on the second of the two farms to which allusion has frequently been made.

Probably some one will give us details of cases where such preventative injections have been made; it would be specially interesting to hear of a case where these have been used and have not been successful in staving off an attack of tetanus. Personally, I feel very confident that there is a great future in store for antitetanic serum, and that in the course of time it will be generally employed as a prophylactic in all cases of wounds from which there is reason to fear tetanus may follow. It is quite certain that with ordinary care as to its use there is absolutely no danger to our patients by injecting it. The syringe and its needle, if made of suitable material, should be boiled before use; this will render it aseptic. Failing that, wash it in water, then in alcohol, and finally in æther.

In addition to injections it behoves us to keep the wounds clean by antiseptic dressings, and to take care that the animal's surroundings are as hygienic as possible. In the present state of our knowledge it would be unwise to allow a box or stable in which a case has stood to be again occupied before a thorough cleansing and limewashing has taken place. I think we may also fairly say that the dressing of pasture lands with the scrapings from roads and dust contractors' refuse is to be deprecated.

As I have opportunities of seeing the process, perhaps a few words as to the mode of preparation of the serum may be of interest.

A young horse in good condition is selected and is tested with mallein and tuberculin to ensure its freedom from glanders and tuberculosis. An artificial cultivation of the tetanus bacillus in broth is grown for four or five weeks; it is then filtered through porcelain, and a very small quantity, about half a drop, of this filtrate is injected subcutaneously into the horse. On the third day the dose is repeated If the animal shows no sign of tetanus about the third day afterwards a whole drop is injected, and for several months the process is repeated with continually increasing doses of the poisonous filtrate until the horse's system is so resistant to the poison of the bacillus that he will take large quantities without ill effects. Thus, at a period of six months after the first injection he will probably be unaffected by a dose of 2 ounces, and at the end of twelve or eighteen months as much as a pint, or even more, can be injected with impunity. At frequent intervals during this process a small portion of blood is taken from the horse, and the potency of its serum is tested experimentally on mice and guinea-pigs. When it is found to confer sufficient immunity the horse is periodically bled by piercing the jugular with a trocar connected by sterilised tubing with a sterilised and sealed glass flagon, the blood obtained is allowed to coagulate, and its serum is separated and filtered for use.

After the blood has once acquired a sufficient protective power to be serviceable, the horse will, without further dosing, continue to supply potent serum for a long but varying period of time, and it is necessary to continually test the supply experimentally.

The serum may be obtained from the vendors either in a liquid or dried state. The latter, which will retain its potency for a long period, is prepared by dessicating the serum *in vacuo*, and for use it must be ground up in six times its weight of distilled water.

Some will be inclined to ask how such serum exerts its protective properties. I cannot answer the question. Certainly I think it is not a chemical antidote, but it is an established fact that susceptible animals adequately protected so as to reach the highest possible degree of immunity from infection with pathogenic bacteria or from intoxications with their chemical products are capable of supplying a serum which injected into other susceptible animals will protect them against like infections and intoxications, and also in some cases have a curative effect. How it is capable of exerting this power seems to be unknown. Various hypotheses have been advanced, and I cannot do better than quote from an article by Dr Kanthack published only last year. He says, "the mechanism of immunity, whether acquired or natural, specific or general, is still hidden from us, and we cannot as yet lift the veil, yet the recent past has revealed so many marvellous facts unto us that we may confidently look to the future for more light" (Allbutt).

Before concluding let me point out that there is no universal method of standardising the strength of serum. We are compelled to accept the word of the manufacturer as to its strength and the dose we should administer. Unfortunately a commercial spirit seems to have pervaded some of the vendors, who advertise in a puffy way worthy of cocoa or soap. It behoves us not to pay too much attention to this, but, bearing in mind that the serum, from the cost involved in its preparation, must be very expensive, buy only from well-known sources.

SOME FATAL CASES OF PNEUMONIA AND PLEURISY IN THE HORSE.

By Veterinary-Major F. Smith, Army Veterinary Department.

THE fatal cases here recorded are those which have occurred in my practice during the last two and a half years; they form the basis of the article on pneumonia and pleurisy published in this Journal (vol. X., Nos. 1 and 2), and the paper in question may perhaps be consulted with advantage when, from the necessarily brief manner in which the clinical records are reported, any point in connection with the chest sounds may appear obscure. All the horses were young, the majority being four years old.

I have drawn certain conclusions from the recorded facts; those having a particular bearing on any special case are inserted under the head of "Remarks." The other conclusions arrived at have appeared in the paper above mentioned.

The value of the present record lies in the fact that it is complete from invasion to death; and, secondly, the facts being correct, the reader is placed in a position to make his own deductions, and is not bound down by those of the observer.

PNEUMONIA.

Case I.

Short History. Admitted with symptoms of fever, but no lung trouble discovered. On the second day the right lung became affected. On the third day the symptoms became very urgent, great depression, grunting, and respiratory distress; hardly any air appeared to enter the left lung, and very little entered the right. On the fifth day very great distress, no respiratory murmur can be detected in either lung, but the chest perfectly resonant from top to bottom; pulse fair. On the sixth day worse; up and down several times during the day owing to distress. Seventh day, died.

Pulse, Respiration, and Temperature. The pulse started at 70, for two days remained at 60, and then rose to 80. A peculiar pulse-respiration ratio was present during the last two days of life, the breathing being 40 to the minute. The temperature rose and fell 2° every twelve hours for four days.

Post-mortem Appearance. The lungs did not collapse on opening the chest. They were removed with difficulty, ***but neither cutting nor pressing them caused collapse.*** The lungs looked mottled in patches all over; these were isolated patches of pneumonia, and on section they were maroon in colour, spongy, and sharply demarcated from the surrounding healthy tissue. The anterior lobes of both lungs were hepatised, and contained abscesses; pleura normal.

Remarks. (1) A very remarkable case. I am puzzled to know whether the lungs recoiled to any extent during life, and cannot explain their non-collapse after death. The peculiar pulse-respiration ratio may have been due to this condition of lung. (2) Note the distribution of pneumonia in patches. (3) Observe that though grunting occurred throughout the case there was no pleurisy.

Case II.

Short History. This horse was admitted with pneumonia of the right lung, followed on the sixth day by pneumonia of the left lung. Throughout the case the chest sounds were indefinite; sometimes the lungs would appear to be clearing, and in a few hours no air sound could be heard. This was repeated two or three times. Throughout the attack the patient grunted persistently; it was quite distressing to hear, but not a trace of pleurisy was found *post-mortem.* The heart impulse was so violent as to mask the lung sounds in that region. The patient had an excellent appetite. Towards the termination of the case there was great respiratory distress. On the twelfth day an abscess was located in the left lung owing to the squeaking and bubbling which existed. None were diagnosed in the right, though a fairly large one was found *post-mortem* in a position where it should easily have been found. The patient lived fourteen days.

Outline of Post-mortem Appearances. The lower half of both lungs consolidated; abscesses plentiful in anterior lobes, and in the posterior part of the middle third of each lung was a relatively large abscess.

Case III.

Short History. This horse was under treatment for strangles, the throat being much swollen, and the abscesses deep seated and giving rise to difficulty in breathing. I did not like the condition of the lungs on the fourth day of the attack, but the respirations were ***never more than twelve,*** and the animal, being intensely nervous, could only be sounded with great difficulty. He could not feed owing to the throat trouble, and nutritive enemata did not keep him alive. He died on the sixteenth day.

Pulse, Respiration and Temperature. The pulse was sixty throughout, the respirations twelve, the temperature generally high, though on the fourth day it touched normal.

Outline of Post-mortem Appearances. Right lung anterior lobe solid and full of abscesses; remaining part of lung normal. Left lung contained a solid patch at the inferior third, and in this was an abscess; the patch was sharply circumscribed by healthy lung tissue. There was no pleurisy. The tissues of the soft palate and pharynx were gangrenous, sloughing, and riddled with abscesses; the latter were also found in the tongue. The guttural pouches were black and contained pus; larynx perfectly healthy.

Remarks. Note that pneumonia was present in spite of twelve respirations per minute. The lung trouble was doubtless due to the strangles organism.

Case IV.

Short History. This case was remarkable in living thirty-six days with acute lung trouble. The patient had no pneumonia when admitted; the symptoms set in in thirty-six hours, both lungs being affected. On the sixth day both lungs were blocked below; there was great grunting but no pleurisy. On the eighth day friction was present in the right chest. On the eleventh day there was an improvement, friction had left the right chest, and the left lung was clearer. On the sixteenth day air was entering both lungs to near the bottom, but it was associated with considerable squeaking, and in the left lung with tubular breathing. On the eighteenth day there was a decided relapse, and there were extensive râles in both lungs of a wheezing, bubbling, cooing, and squeaking character. On the twentieth day the patient was worse, there was a harsh respiratory murmur and fine tubular breathing in the left lung, while in the right lung there was marked grunting bronchophony behind the elbow. On the twenty-fifth day there was great muscular wasting in spite of the excellent appetite the patient maintained throughout. At this time both lungs were again dull below on percussion, with an entire absence of air sound. Above the consolidated area there were squeaking crepitations, both fine and coarse. On the twenty-ninth day the dulness on percussion of both chests had nearly gone, the sound being remarkably resonant. Gradually the case got worse, the breathing more distressed, constant grunting, great muscular wasting, and death on the thirty-sixth day.

Outline of Post-mortem Examination. Both lungs consolidated below, abscesses in the anterior lobes, and many abscesses scattered throughout the two lungs, especially the upper half; one of these was very large, caused by the union of several smaller ones. Only a trace of the pleuritic patch was found in the right chest, the remainder of the pleura being absolutely normal, though the patient grunted persistently for over a month. The lungs could not be inflated after death owing to a leak.

Remarks. Note (1) That the lungs commenced to break down on the sixteenth day. (2) Between the eleventh and twenty-fourth days there appeared to be a somewhat clearer condition of lung, and then the organs once more became consolidated. This has been observed in other cases. (3) The pulse-rate, respiration, and temperature indicated that the condition mentioned in (2) could not be real. (4) The resonant chest a week before death was due to pneumo-thorax. (5) Most abscesses were found where there had been the least pneumonia.

Case V.

Short History. Admitted with a high temperature, tender throat, and dribbling of saliva from mouth; the chest remained clear for two days, and then pneumonia of the lower third of the right lung developed. The throat became worse, swollen, and indicated the formation of strangles abscess. On the ninth day one burst internally and discharged through the nose. The appetite throughout was bad, and the patient had to be hand fed; the left lung remained clear, and the amount of trouble in the right was never very extensive. The patient died on the thirteenth day. Both pulse-rate and respiration were persistently high during the whole of the attack.

Outline of Post-mortem Examination. The lower third and anterior lobe of right lung consolidated and full of abscesses. Great destruction of the tissues of throat and tongue, deep-seated abscesses in the latter, sloughing and intense thickening of the soft palate, pharynx, and epiglottis; the soft palate was at least 1 inch thick; the submaxillary glands were full of pus.

Remarks. The pneumonia in this case was probably due to the strangles organism.

Case VI.

Short History. The peculiar feature in this case was the doubt that existed for some days as to whether pneumonia were present or not, the chest being naturally an extremely dull one, and the patient one of the few met with where no deep inspiration could be produced. It is curious that for days I should have remained in positive doubt as to the existence of pneumonia, and even when extensive consolidation of both lungs was definitely ascertained the other symptoms present did not support it. Note particularly the pulse and respirations. It was not until the seventh day after admission that I was beginning to feel anxiety about the lungs, and during the whole of this time the respirations were only 14 per minute. On the fifteenth day the patient was brighter, better, and feeding well. On the eighteenth day there was a relapse, and on the twenty-fifth day he died.

Pulse, Respiration, and Temperature. The pulse for the first and second weeks was natural to the attack, but during the third and part of the fourth week, at a time when extensive changes had occurred in the lungs, it was only between 40 and 50 beats per minute, and of good character; it only reached 60 and 80 shortly before death. The temperature rose and fell in a very erratic manner; it was only one degree above normal on the seventh day, when the nature of the attack was becoming manifest; it reached normal on the fourteenth and fifteenth days, and was only one degree above normal from the fourteenth to the eighteenth day; it was normal on the nineteenth and twentieth days, then rose again during the last week of life. This temperature curve is most unusual and very misleading, especially when taken in conjunction with the respirations. These were *from* 12 *to* 16 per minute during the entire attack, rising to 24 the day before death.

The Chest Sounds were, as previously mentioned, very indefinite. On the eighth day there was extensive consolidation of both lungs. On the thirteenth day the area of consolidation could be mapped out by the tracheal reflex method. On the twentieth day the left lung was still solid below, but the right was admitting air to the bottom, though the murmur was decidedly of an oppressed character, and five days later this right lung was again found solid. This feature will be apparent in more than one of the cases recorded in this series. I can offer no explanation of the circumstance.

Outlines of Post-mortem Examination. A very large abscess containing necrotic tissue in the middle of the right lung, multiple abscesses in the anterior lobe, and considerable consolidation of the inferior third. In the left lung there was *no abscess*, the anterior lobe was normal, but there was extensive hepatisation of the inferior lobe.

Remarks. This was a very misleading case. (1) The physical examination of the chest, respirations, and temperature gave no assistance in determining the nature of the attack in the early stages, and neither respirations nor temperature gave any help in prognosis throughout the entire case, while the pulse-rate of the third week was positively misleading. (2) The single lung abscess was the largest I have seen. I did not determine its existence during life, though its position was quite favourable for diagnosis. It could have held about half a gallon of liquid. (3) It is interesting to notice that that most fatal of places for multiple abscesses, viz., the anterior lobe, was quite normal in the left lung, and further, that there were no abscesses in any part of this organ.

Case VII.

Short History. This horse was admitted off feed, the pulse and respirations being normal, the temperature 102°, the chest clear. The temperature steadily rose up to the fifth day, at which time changes in the lung began. By the sixth day pneumonia of the right lung was established. On the eighth day the left lung became affected. The case lived for twenty-seven days.

Pulse and Respirations. During the first week the pulse was from 40 to 48, the breathing frequently only 12 or 14. During the second week the pulse was generally 60, while the respirations were never less than 30. During the third week the pulse was generally 48 and the respirations 24, while in the last week of life they were as high as during the second.

Chest Sounds. The chest sounds were as follows. On the day when the lung became suspected there was a modified or oppressed respiratory murmur below, the air was not entering as freely as it should do, there was an increased murmur in the healthy portion of lung; on deep inspiration there were no crepitations, and the oppressed respiratory murmur caused by the deep inspiration rapidly died away. On the day pneumonia became firmly established, viz., the sixth, hardly any air was entering the affected lung, no crepitations, but occasional expiratory squeaks. On the eighth day the left lung became affected like the right, excepting that bronchial breathing was already found. On the ninth day there was a remarkable extension upward of the lung trouble; the bottom of both organs was absolutely solid, only faint squeakings being heard even at deep inspiration; the bronchial breathing had gone from the left lung. Tenth day, both lungs absolutely blocked for two-thirds, and yet the percussion note was remarkably resonant, excepting at the lower third. No bronchial breathing or tracheal reflex present. On the thirteenth day both coarse and fine squeakings were heard over the middle of the right lung; the squeakings were both inspiratory and expiratory, but generally inspiratory. On the fourteenth day the squeakings were heard all over the lower third of both chests; these squeaks and coarse bubbles were very marked about the centre of each lung, and were present during both inspiration and expiration. Swallowing bronchophony could also be heard. Twenty-third day, the râles described on the fourteenth day were now more marked and coarser, resembling croaking and grunting; no bronchial breathing, and the chest quite resonant on percussion in spite of the complete consolidation of the lower half of each lung. Twenty-fourth day, air entering all over the left chest, a remarkable change from yesterday. No air entering the lower two-third of the right chest, and the grunting, bubbling, and squeaking râles over the middle of the lung were marked. These places are regarded as the site of abscesses. Both chests clear on percussion, but swallowing bronchophony can now be heard. Twenty-fifth day, air still entering the left chest, but at first part of upper third coarse râles were heard both at inspiration and expiration, and there were loud moist crepitations in this region. It is believed that this lung is breaking down. The squeaking râles in the right chest are still present, and in addition loud moist crepitations over nearly the whole of the lung. Twenty-sixth day, the left lung contains only one affected area, and here the râles are converted into coarse bubblings, heard both at inspiration and expiration. In the right chest the entire lung is practically occluded, loud liquid bubblings are heard over the area previously indicated as necrotic, and grunting bronchophony is now present. Twenty-seventh day, found dead in the box, the place covered with blood, owing to hæmorrhage from the lungs, which evidently was the immediate cause of death.

Outline of Post-mortem Appearances. The anterior lobes of both lungs riddled with abscesses; abscesses over the middle third of right lung, where indicated during life, many of them situated in the middle of comparatively healthy lung tissue; another large abscess high up at the posterior part of the lung which was not detected during life. Both lungs at their inferior third were hepatised. The blood-vessel had burst in one of the pus cavities in the anterior lobe of the left lung. Pleura normal.

Remarks. Note (1) The delay in the onset of pneumonia. (2) The low pulse and respiration-rate during the first week. (3) The delay in the attack of the right lung. (4) The remarkably clear percussion note. (5) The commencement of the lung necrosis on the fourteenth day. (6) The extraordinary change in the left chest on the twenty-fourth day, unexplained by *post-mortem* examination. (7) The rupture of the pulmonary vessel. (8) The unusually long period the patient lived.

Case VIII.

Short History. This horse on admission presented no definite condition indicating pneumonia, but in thirty-six hours these appeared, both lungs being affected. The patient fed well. The case lived nearly thirteen days, and for the last three days of life was in great respiratory distress, getting up and down constantly during this time. I never before saw such long continued distress.

Pulse, Respirations, and Temperature. The pulse and respirations during the first week were normal to pneumonia—a pulse of 60 and respirations 30, but during the second week the pulse-respiration ratio was very curious, viz., a pulse of 72, and respirations of 60, or a pulse of 86, and respirations of 74. The temperature on admission was 107°, with a fall to normal on the morning of the second day, but with a rise in the evening to 104°. I have observed this drop to normal and subsequent rise on more than one occasion.

Chest Sounds. First day, oppressed respiration. Second day, absolute dulness of both lungs, lower third. Third day, extension of disease upwards; a deep inspiration producing wheezing in the affected area. Sixth day, squeaking râles are heard at intervals all over the chest. Seventh day, bronchial breathing behind left elbow. Tenth day, over the middle of the right lung is an area of intense squeaking râles, heard both at inspiration and expiration; very little bubbling, though some is present. I regard this as an abscess forming. In the left lung is a large area of bronchial breathing behind the elbow. Eleventh day, increase in the squeaking and grating area in the middle of the right lung, and tubular breathing below. Considerable squeaking and cooings in the left lung at different parts. Twelfth day, bubbling and squeaking râles as before, hepatisation of both lungs below, tubular breathing in both chests behind the elbow.

Outline of Post-mortem Appearances. Both lungs contained innumerable abscesses in the anterior lobes, the lower third of both lungs firmly consolidated, and where the squeakings and bubblings were heard during life were large abscesses, many of which were some distance from the inflamed area. An attempt to inflate the lungs after death failed on account of a leak into the pleura.

Remarks. Note (1) the strange pulse-respiration ratio during second week. (2) The sudden drop in the temperature on second day. (3) Commencing necrosis of lung on the tenth day.

PLEURO-PNEUMONIA.

Case I.

Short History. Admitted with sore throat and high temperature; on the fourth day pneumonia occurred, the pulse went up to 80, the respirations to 36. With very little variation the symptoms lasted two weeks, the case dying on the nineteenth day after admission. Effusion occurred as early as the third day following the acute symptoms.

Outline of Post-mortem Examination. Revealed abscesses in both lungs, abundant putrid effusion, lungs adherent superiorly to chest wall and freely plastered with lymph.

Case II.

Short History. This was an extremely rapid case, admitted one evening with a temperature of 104°, pulse 45, respirations 20. I did not see the patient on admission, so do not know the state of the chest. The following morning there was marked friction over left lung, great collapse, hardly any pulse, which was 60 to the minute, short abdominal respirations 36. On the third day the case was dying, the pulse 80, respirations 48; in the afternoon the patient got down and was unable to rise even with assistance. He showed signs of what appeared to be abdominal pain, was pulseless, and even the heart sounds could not be distinguished. Convulsions occurred at intervals, and he died the same night, fifty-two hours after the attack.

Outline of Post-mortem Appearances. Effusion of blood-coloured serum into the left pleural sac only, membranes on the lung but none on chest wall. Left lung inferior third hepatised. Right lung congested but otherwise normal. Pericardial sac over an inch in thickness from serous exudation the result of inflammation, and there was some fluid within the sac.

Remarks. (1) A curiously sudden case. Why the collapse? The right lung was perfectly fit to carry on the needful work for a long time, and this sudden case is very suggestive of a poison. (2) There was no abscess in these lungs, nor indeed should we expect such within so brief an illness. (3) The effusion was not diagnosed during life, and it is one of the very few cases met with where the exudation was single sided.

Case III.

Short History. This horse was found ill with a temperature of 106°, a pulse of 100 which could not be taken at the jaw, and even the heart-beats indistinct; the breathing 54 to the minute, but strange to say, in spite of this rate, there was not the slightest movement of the nostrils. The chest was quite clear. On the second day there was well-marked pleuritic friction of the right chest, great distress and grunting, and heart sound still very muffled. On the third day the patient fed well, friction still present over right lung, and off fore leg advanced. On the fourth day there was friction over both left and right lungs, and pericardial friction likewise. On the sixth day feeding well, friction sound over both chests gone, and now it is possible to ascertain that both lungs are consolidated below. Ravenous appetite. Seventh day, not so well. Eighth day, no improvement, off fore leg still advanced. Ninth day, worse, great respiratory distress, for which the pleural sac was tapped, but only a little fluid came away. Knowing the pericardium to be affected, and thinking the respiratory distress might be cardiac, the pericardial sac was tapped, but obtained only a little fluid, and on pushing the instrument in deeper obtained an alarming gush of blood. Tenth day, great respiratory distress with dilated nostrils, anxious expression, air entering to the bottom of right chest, which made one think there could be no effusion present, but in this, as the *post-mortem* will show, I was mistaken, Refuses all food. Tenth day, died.

Pulse, Respirations, and Temperature. The pulse started with 100, fell on the fourth day to 48, then rose again, and was at 100 the last two days of life. Though very oppressed at first, it subsequently became of much better volume. The respirations throughout were between 50 and 60. The temperature was very high, 106° being reached three times.

Chest Sounds. While the friction lasted it was very intense; it was of a cracking, creaking character as heard through the phonendoscope, and masked all the lung sounds by its intensity. It is seldom that the sound is so intense.

Outline of Post-mortem Appearances. Abundant effusion into both chests, great collapse of both lungs from hydrostatic pressure. The lower half of the chest was plastered with lymph, and the inferior half of the lungs was in a similar condition. The inferior fifth of the right lung and rather less of the left were pneumonic and consolidated, and on cutting into this portion it was found full of abscesses. The remaining portion of lungs was healthy. The diaphragm was coated with lymph from top to bottom. The pericardium enormously thickened and blood stained, while the sac itself held about a pint of blood-coloured fluid. There were two marks in the left ventricle near the apex, indicating where the trocar had touched it, but neither had penetrated the wall of the ventricle, at which I was surprised, considering the stream of blood which came through the canula.

The horse was a whistler, but the larynx showed no disease.

Remarks. (1) This horse died from slow asphyxia, due to the accumulation of fluid in the chest. I probably could not find the latter owing to the membranes present. (2) Air appeared to be entering to the bottom of the chest on the ninth day, at a time when the lungs were not within several inches of the sternum. The sound is therefore transmitted through the fluid in certain cases, and is *most misleading.* (3) The pericardial tapping is not a very satisfactory record, but fortunately did no harm. (4) The high pulse and respiration rate of this case is noteworthy. (5) Pointing one or both fore legs is not uncommon early in pleurisy.

Case IV.

Short History. Admitted with a temperature of 106°, pulse 70, respirations 44. The patient walked to the infirmary, only a few yards distant, and was almost prostrate, so intense was the sudden depression. The animal grunted, but nothing definite could be made out of the lungs until the evening, when it was evident both were affected. Heart sounds were feeble. On the second day there was great collapse, almost pulseless; temperature in the evening 107°, pulse 100, respirations 48. Third day, pulse 100, respirations 40, temperature 105°. In the left lung little or no air is entering below, and where it is cooing is present. The right lung is fairly clear. Died sixty-two hours after admission.

Outline of Post-mortem Examination. The whole pleural membrane of both sides minutely injected and ecchymosed, but no membranes. The pericardium was a sight, simply one big splash of blood extravasation, beneath which was a thick gelatinous effusion. The left lung was pneumonic, especially *superiorly* (which is rare), and possessed the same marbling as a pleuro-pneumonic lung; in the left sac was a little fluid. The right chest contained volumes of blood-stained fluid, the right lung pneumonic below and hepatised, and there was also marbling of the superior lobe of the lung, as in opposite chest. The heart was quite extraordinary; it was speckled with blood spots, the fat gelatinous, and the pericardial sac contained much fluid. The trachea and bronchial tubes were nearly black, and even the tissue *outside* the trachea œdematous. In the pharyngeal glands was some creamy pus, while the guttural pouches were purple.

Remarks. (1) A very virulent case affected from the fauces to the diaphragm. (2) The effusion was undiagnosed, and it will be noted that air was heard entering to the bottom of the right chest on the last day of life, though *post-mortem* examination showed that no air could have entered. (3) The heart lesions were curious, during life the sounds were extremely feeble.

Case V.

Short History. Admitted with pleurisy and pneumonia, the physical signs of the disease being very marked; there was an entire loss of appetite, and very rapid thumping action of the heart, and in this way the case continued up to the tenth day, when some improvement in the symptoms was manifest; on the sixteenth day there was a distinct relapse, the breathing indicated effusion, and the chest was tapped; already the fluid had a faintly fœtid odour; on the nineteenth day he died.

Pulse, Respirations, and Temperature. Nothing to record, quite normal to the case. The pulse was at times peculiar in character, some beats being full and good, and others being so indifferent that the artery appeared almost empty. The pulse was occasionally intermittent. The heart's action against the chest wall was most violent from the first.

Chest Sounds. On admission well-marked pleuritic friction of the left chest and dulness of left lung below; no friction in right chest, but lung dulness well marked below. On the second day the consolidation of the lungs had increased, still friction over the right lung, but none over the left. On the fourth day the friction had disappeared, a little air was entering the left lung below, but none in the right. On the fifth day no air could be heard entering the left lung below, the right still solid, fine tubular breathing could be heard in the left lung for the first time. On the thirteenth day bubbling was heard over the larger bronchi on the left side, and tubular breathing below in the right lung; no respiratory sound whatever below. On the fifteenth day tubular breathing appeared in the right lung at the bottom, left lung no change. On the seventeenth day, though abundant effusion existed in the chest, tubular breathing could be heard at the bottom of the chest in the entire absence of lung, showing that this sound was transmitted through the fluid. The abdominal breathing was characteristic of effusion.

Outline of Post-mortem Appearances. Pleurisy of the lower half of the chest wall and the whole of the pulmonary surface; not a single false membrane, the pleura being simply scarlet and maroon. Anterior lobe of right lung turned back, and glued to the outer surface of the upper third of the lung. This right lung was carnified from hydrostatic pressure, and contained a large abscess close to the bronchi, but I could see no pneumonia; the right lung was also attached to the ribs. The left lung was attached to the chest wall and to the diaphragm; it contained in its interior an abscess like the right lung, was carnified from pressure, and had scattered through it islands of pneumonic patches, more in the form of hæmorrhages, but no attempt at hepatisation. The pericardial sac was black and scarlet, and greatly thickened, but no false membranes.

Remarks. (1) There are some peculiar features in this case; one important one is the absence of membranes in acute pleurisy; none were found anywhere, not even in the fluid from the chest. (2) Note the violent action of the heart and the pulse peculiarities, such are not uncommon as the result of the pericarditis. (3) Observe that there was not a single patch of hepatisation in these lungs, and that one at least would have been normal had it not contained an abscess and become carnified from pressure. (4) Note that respiratory sounds were heard where no lung existed, and also that the solid lung I expected to find did not exist.

Case VI.

Short History. On admission this horse was found to have pleurisy on the left side, the pulse 92, could hardly be felt, and the heart ticking like a clock. The lungs appeared clear, at least a normal murmur could be distinctly heard through the friction. Second day, pulse better, right chest still clear. Third day, friction gone from left chest, but the lung is now affected, the respiration being very oppressed; right chest not quite satisfactory. Fourth day, marked respiratory distress, feeding badly, extension of lung trouble. Fifth day, there has been a peculiar pulse-respiration ratio, which is extremely marked now, viz., a pulse of 50, and respirations of 40. The chest is admitting more air through the affected portion. Sixth day, better, feeding, and brighter. Eleventh day, has been doing well up to to-day, when the appetite has failed; the right chest is extremely wooden on percussion, and no air entering its lower half; air enters the left lung, but very imperfectly. Thirteenth day, evidently doing badly, great loss of condition, improvement in pulse, peculiar ratio still present, chest sounds no change. Sixteenth day, the respirations are characteristic of effusion, bronchial breathing present in right chest behind the elbow. In the left chest air can be heard to the bottom, yet the chest contains a considerable amount of fluid as proved by tapping. Seventeenth day, died.

Pulse, Respirations, and Temperature. The pulse was generally 60, excepting the first and last days of life, when it was 90. The respirations were between 30 and 40, so that the pulse-respiration ratio was very singular; for example, a pulse of 50 and respirations of 40. The temperature was seldom high, and on the tenth day reached normal.

Outline of Post-mortem Examination. Only the lower half of the chest affected with pleurisy, the upper part quite free; diaphragm affected. No membranes of any kind found in the chest, but the affected pleuræ were engorged, even in some places to blackness. The right lung was pneumonic below, but the left was free; both lungs were greatly compressed from hydrostatic pressure, and carnified in consequence. In the left lung, close to the bronchus, was a large abscess, but the surrounding lung tissue did not look pneumonic.

There was perforation of the pleura, so that the lungs could not be distended. The pericardial sac was thickened and quite black externally, and even the internal surface was slightly affected, which is very rare. Both lungs and diaphragm were adherent to the ribs in many places.

Remarks. (1) This case presents a feature very common in hydrothorax, viz., a decided improvement in the symptoms followed by a relapse. (2) The pulse-respiration ratio is very curious; it cannot be accounted for. (3) Observe, again, that air appeared to be entering to the bottom of the chest on the last day of life, though the thorax was full of fluid.

Case VII.

Short History. This horse was admitted with a temperature of 104°, no disturbance of the breathing and but little of the pulse. The chest was perfectly clear until the evening of the fourth day, when a little squeaking was heard behind the left elbow, though the air was entering the lung, and the pulse, respirations, and temperature were nearly normal. On the fifth day the temperature rose 4°, and on the evening of this day undoubted lung trouble was present, both sides being affected, and strange to say the upper part of the lung was more involved than the lower—one of the very rare exceptions met with. The pulse was good, the respirations of a peculiar snorting type not uncommon in acute chest trouble, and the near fore leg was advanced, resting on the toe. Eighth day, no change, pulse particularly good. Tenth day, worse, grunting, and painful cough, extension of disease in both lungs, no appetite. Twelfth day, worse, great depression, grunting, no appetite, but pulse wonderfully good. Fourteenth day, a pleuritic patch discovered, patient much worse, pulse failing. Sixteenth day, no change, snorting respiration still present; it is due to the false nostril being drawn in at inspiration. Eighteenth day, still alive, pulse a mere thread, slight blood-stained discharge from the nostril. Nineteenth day, died.

Pulse, Respirations, and Temperature. During the period of incubation the pulse and respirations were normal; later the pulse rose to 60, respirations to 24, and so remained until a few days before death. The temperature on admission was 104°, and then fell to nearly normal, until the fifth day when it rose to 105°. On the seventh and eighth days it again fell to nearly normal, and never subsequently rose above 103°.

Chest Sounds. On the fifth day both lungs dull in the upper third, squeaking behind right elbow, and no air entering behind the left. Sixth day, chest clearer than on fifth, but the patch of consolidation in the right lung is still present, and on deep inspiration there is squeaking and crackling. Seventh day, left chest clear. Right chest, undoubted pneumonia both behind the elbow and above; pneumonic crackling on deep inspiration and much wheezing. Ninth day, the left lung is now affected, and like its fellow is attacked both above and below. Tenth day, both lungs blocked below for more than one-third, squeakings can be heard on deep inspiration, more marked in the right than the left lung. Bronchophony in left and not in right lung. Thirteenth day, pleuritic friction behind right elbow, no air entering either the top or bottom of the lungs, the middle portion only carrying on the work. Fourteenth day, tubular breathing in left chest below, and grunting bronchophony; tracheal reflex well heard. Extension of friction behind the right elbow. Eighteenth day, left lung completely blocked above and below, yet on percussion the note above is clear, though no air is entering the lung. In the posterior portion of this upper region is a part where at each inspiration a peculiar sound is heard, such as might be produced by dropping a stone into a well. On the chest chart I marked this as probably an abscess, though I had never heard the sound before. *Post-mortem* examination showed that there was no abscess. In the right lung the lower third was solid; above this an area of fine squeaking bubbling crepitations. In the upper-third were also crepitations.

Outline of Post-mortem Appearances. The lungs could not be inflated, showing perforation had occurred. Right lung anteriorly was sold from top to bottom, the only breathing area was the upper and posterior part; no abscesses. Left lung one solid mass, though not so firm as the hepatisation of the right lung; in the anterior lobes were innumerable abscesses. Marked pleuritic roughness over right lung and chest wall, but no fluid in the chest.

Remarks. (1) This is a very exceptional case; it is seldom that an entire lung is destroyed by pneumonia, and certainly the left was in this patient. (2) It is curious that the abscess production should have been so limited.

Case VIII.

Short History. This is an extremely interesting case from the rapidity with which the disease ran its course, killing the patient in three days. This horse on going out to exercise one morning was observed to be unusually fit and well. The exercise consisted of walking for two hours at quite a moderate pace; the animal returned from it in such a state of collapse that it was with difficulty it could be dragged to a loose-box, and this will be readily appreciated when it is stated that the temperature was 108°, the pulse 108, and the respirations 40. There was the most intense depression, the left chest presented one small patch of pleurisy, and the respirations below were of an oppressed character. In the right lung the inferior third was partly blocked, and pneumonia present. This condition of the chest was in itself insufficient to account for the very acute character of the symptoms. On the second day he was obviously dying, and there was a large pleuritic patch at the upper and posterior third of both lungs; great consolidation of right lung for more than one third, and well-marked changes occurring in the corresponding part of its fellow; in addition the patient was shivering and blood coming from the nostrils. On the third day the pulse was 128, counted at the heart, nothing could be taken at the jaw. The chest symptoms remained unchanged, excepting that there were marked fine crepitations over the middle of the left lung. There was great distress, pawing with the fore legs, resting the head anywhere, against the wall or the manger, moving round the box and unable to see where he was going, and other symptoms pointing to delirium. Occasionally there would be severe attacks of dyspnœa, the tongue would be protruded and become livid, and breathing would take place through the mouth; these symptoms passed off as rapidly as they came on. The breathing did not indicate effusion, though I might have suspected it from the respiratory distress, but I hardly expected any serious effusion in three days. He died on the third day.

Pulse, Respirations, and Temperature. The pulse on admission was 108, it never fell lower than 80, and was more often 100. The breathing never fell below 36, and was 48 before death. The temperature of 108° on admission in the morning fell to 106° at 5 P.M., and 104° at 8 P.M. On the second day it again rose to 106°.

The Chest Sounds were not very satisfactorily made out; the lung which appeared the least affected turned out on *post-mortem* examination to be the worst, and again I met with that feature which I cannot too frequently record, viz., at one examination and for a brief time one lung appearing *comparatively* clear, though at the previous examination a few hours before it was blocked below, and the same condition found a few hours later. Were it not that I have met with this curious feature many times I would put it down to an error of observation. It only shows that we cannot examine the chest too frequently. During the last few hours of life air could be heard entering to the bottom of the chest though the cavity was full of fluid. I am noting this condition wherever in this series it has been observed, as the symptom has its practical bearing—the respiratory murmur is transmitted by the fluid.

Outline of Post-mortem Examination. Both chests full of blood-coloured serum, containing clots of fibrin-like curds. Acute pleurisy of lung, chest wall, and diaphragm. Left lung just behind the heart was swollen and firm, and the mass of a greenish colour, indicating gangrene. On cutting into it, it proved to be little else than one vast blood clot due to hæmorrhage; above the apoplectic area the lung was greyish and pneumonic. The whole of this diseased condition was almost entirely limited to the inferior third of the organ. The right lung was affected with red hepatisation but no apoplexy. No abscesses could be discovered in the lungs, and the anterior lobes were normal, which is very rare. The lungs distended under inflation. The pericardial sac was enormously thickened and discoloured from inflammation.

Remarks. (1) Note the rapidity of the invasion and the rapid progress of the case. (2) The intensity of all the symptoms excepting those of the chest, which though severe were not sufficiently so to account for the collapsed state of the patient. (3) It is possible that cases of this sort are different from ordinary pneumonia. (4) It would certainly appear to be a virulent poison which can affect such a relatively small area, and cause such urgent symptoms and death.

Case IX.

Short History. Admitted in the evening "blowing" and occasionally lying down. The pulse peculiar, swells under the finger, then dies away; marked laryngeal respiration; both lungs were affected and the pleura on the right side. On the third day the pulse was excellent, feeding well, temperature and pulse dropped, and every indication of improvement. Everything went on well, temperature, pulse, and respirations normal until the ninth day, when the temperature rose, and all the old symptoms returned; from this to the thirteenth day there was a gradual increase in all the symptoms, and death then took place.

Chest Symptoms. On admission both chests below were very oppressed, and on deep inspiration there were crepitations. On the second day the chest was clearer and no crepitations, but on the evening of that day well-marked pleuritic friction existed; this had disappeared the following day, and only an extremely oppressed respiratory murmur remained. On the ninth day there was a relapse; the area of lung dulness became much greater, there being no respiratory murmur below even on deep inspiration. Tenth day, tubular breathing present behind left shoulder, other symptoms as before. Eleventh day, the dulness on percussion is intense, but the respirations do not indicate effusion. Swallowing bronchophony present in left chest. Twelfth day tubular breathing all over the bottom of the left chest (though *post-mortem* examination showed it to be full of fluid), respiratory dulness everywhere, pleuritic friction above. In the right chest pleuritic friction above, entire absence of any murmur below, no tubular breathing (yet this chest was full of fluid as on opposite side). Thirteenth day, tubular breathing all over the bottom half of both chests.

Outline of Post-mortem Examination. Both pleural sacs from top to bottom and front to rear were plastered all over with fibrinous lymph. There was an abundant putrid effusion, entirely unrecognised during life though repeatedly looked for. The right lung was pneumonic at the inferior third behind the heart; it contained two large abscesses. The left lung though covered with lymph was perfectly free from pneumonia. The anterior lobes were quite healthy. The collapse of the lungs from hydrostatic pressure was remarkable. The percardial sac was enormously thickened from inflammation.

Remarks. (1) Note the relapse after what appeared to be complete convalescence. (2) Tubular breathing in spite of effusion. (3) The freedom of the right lung from pneumonia in spite of the acute pleurisy.

Case X.

Short History. Admitted with well-marked pleuritic friction of the left chest, with an exceedingly bad pulse of 60, high temperature, but respirations only 14. On the second day the friction was still present, and through it the respiratory murmur could be heard; behind the heart the friction was almost continuous, due to pericarditis; in the right chest behind the elbow was a dull patch. Third day most intense friction in left chest, at times almost crackling; there is a small friction patch behind the right elbow. There is no appetite, the temperature higher, pulse 72, and respirations 24. Fourth day, in the evening the friction had disappeared. On the fifth and sixth days there was a decided improvement, pulse and respirations fell considerably, but no important fall of temperature. On the seventh day there was a relapse, the pulse and respirations rose; the left chest was very dull on percussion, and tubular breathing was present, while in the right chest there was increasing dulness behind the elbow. On the eighth day the lower half of the left chest gave a very oppressed murmur, and in addition there were very coarse harsh râles present and also grunting. On the ninth day there was a blood-tinged discharge from the nostrils and fœtid breath. The appetite had been satisfactory since the fifth day, but had now failed. Eleventh day, fœtid grumous discharge from nostrils. The chest examination revealed the inferior part of the left chest *clear* on percussion; and occupying the middle and lower third of the lung was an area full of moist râles something like the bubbling of a boiling pot. It is considered that this is due to an abscess; in the right lung there is a place behind the elbow where bubbling is occurring, and this is also regarded as an abscess. Twelfth day, died.

Outline of Post-mortem Examination. Both pleural sacs and diaphragm intensely inflamed. A very large abscess existed in the left lung where located during life, and it freely communicated by numerous holes with the pleural sac; the lungs consequently could not be inflated. The right lung was one-third consolidated, but contained no abscess. The bubbling heard on this side must therefore have been reflected from the opposite lung. The pericardium was severely affected.

Remarks. A *single large* abscess (probably resulting from the fusion of several small ones) in the lung is very rare

PLEURISY.

Case I.

Short History. Admitted very dull, temperature 105°, pulse 80, very oppressed and intermittent, respirations 25. No friction on auscultation, but there is almost entire absence of air from the base of the lungs. On the fourteenth day effusion occurred, but, unlike what generally takes place in these cases, there was no indication of improvement prior to effusion.

On the fourth day the temperature fell to 102°, but the pulse and respirations were still high, though the volume of the latter had improved. The temperature taken as a whole was high throughout the attack, it being nearly always 104°. The pulse was peculiar; after the first day or two it became large and full, and remained so throughout the attack. The breathing was uneven, short or catchy, and the cough was strong, the latter symptom being misleading and unusual.

Outline of Post-mortem Examination. General pleurisy of both sides—lungs and ribs. The lungs were collapsed from hydrostatic pressure, and were free from disease if we except an abscess between the bronchi, which I think was only a gland and actually outside the lung. The collapse of the lungs was remarkable; there was practically no lung below a horizontal line drawn on a level with the base of the heart. The pericardium was acutely inflamed and thickened. There was aneurism of the mesenteric artery (parasitic), which probably accounted for the pulse during life. There was an abundant exudation in the chest; it was turbid but free from odour.

Case II.

Short History. This horse had been standing in a railway siding for some hours on a very cold day, and in the evening he was attacked with a shivering fit. The chest was dull, but there was no friction, the temperature 106°, pulse 60, respirations 36. The breathing was short and painful, and later on grunting. The case did well for a few days, temperature falling to 102°, the pulse to 40, and the appetite being enormous. On the seventh day there was a relapse, but there was no indication of effusion. The pulse was good, and the appetite remained excellent. On the thirteenth day the respirations indicated effusion, the chest was tapped and a putrid fluid obtained; the pulse was 100, the respirations 36. He died on the sixteenth day, feeding to the end.

Outline of Post-mortem Appearances. The entire pleural sacs were covered with dense membranes, and superiorly the lungs were adherent throughout their entire length to the ribs, and had to be dissected away. The lungs were also adherent to the diaphragm. The lungs were perfectly healthy, no trace of pneumonia; the inferior lobes were collapsed and carnified from hydrostatic pressure, but the organs were quite healthy. Careful search was made for an abscess to account for the putrid condition of the chest fluid, but none was found. The pericardium was intensely affected; there were many gallons of putrid effusion in the chest.

Remarks. (1) Observe the relapse on the seventh day. There is no doubt that at this time effusion commenced, though it was not recognised until later. (2) The entire freedom of the lungs from disease shows that cases of pure pleurisy are possible, though very rare. (3) I never before or since saw a putrid effusion in the chest which was not due to infection by an abscess.

Case III.

Short History. Admitted with intense pleuritic friction over the left chest, and a smaller patch behind the right elbow. It was the most intense friction I have ever heard, and over the region of the heart was synchronous with the heart-beats, showing that pericarditis was also present. The temperature was high, and the horse very ill. Second day, friction still present. Third day, friction gone, chest excellent; a modified though distinct murmur can be heard over the base of the lungs, appetite poor. Fourth day, friction returned in left chest, while the right chest was very dull behind the elbow. Up to the ninth day the case appeared to make fair progress, but the temperature was very variable, and though the pulse did not fall below 50 the respirations dropped to 12. On the tenth day the dulness of the chest was found to extend half-way up on either side, and though the breathing did not indicate it effusion was suspected, as the line of dulness was very sharp, and œdematous swellings of the sheath and brisket were present—a fairly constant symptom in hydrothorax. Fifteenth day, has been doing fairly well up till to-day, but now very dull. The chest sounds on percussion are of a wooden character, and no air enters the lower half of both lungs; the breathing does not indicate effusion. Seventeenth day, relapse, greater distress in breathing, which has risen from 14 to 25, so tapped the right chest and drew off 5 quarts of serum. Eighteenth day, tapped left chest as the breathing was still distressed; drew off 6 quarts, and relief was obtained. Twenty-first day, tapped both yesterday and to-day, 10 quarts removed; tubular breathing is present over the lower third of each chest, while in the right chest superiorly there is a very oppressed respiratory murmur. Twenty-fourth day, feeding well, the tapping of to-day had a faint odour, the breathing does not indicate hydrothorax. Twenty-seventh day, slight improvement in general symptoms, tapped left chest to-day, and the respirations were somewhat deeper, the fluid from this side was sweet. Thirty-fourth day, by this date both chests contained fœtid fluid, but the patient has managed to hang on in the most remarkable manner; there is also a nasal discharge with a fœtid smell. Died.

Temperature, Pulse, and Respirations. The temperature started at 106°, and for the first week there were considerable daily variations, as much as three degrees between the morning and evening temperature. During the second week the temperature fell, reaching normal on the fourteenth day. It rose on the seventeenth day owing to the relapse, but during the fourth and fifth weeks of the disease it remained very little above normal. The only point in connection with the respirations is that for the first two weeks they varied from 12 to 14, which is extremely low for acute pleurisy. On the fourteenth day, with a temperature of 100°, pulse 48, and respirations 12, one had every reason for believing that recovery would possibly follow.

Outline of Post-mortem Examination. Both lungs collapsed to an extreme degree; neither of them extended below the base of the heart. The inferior half of the chest was filled with putrid fluid. The right lung was firmly fixed to the ribs above by adhesions, carnified from compression, and contained anteriorly one abscess. The left lung had no membranes upon it, and with the exception of a little pneumonia along the lower edge and a small abscess it presented nothing of importance; it was of course considerably reduced in size by the compression to which it had been exposed. The pericardium was enormously thickened. At least one of the bronchial lymphatic glands was in a state of suppuration. The lungs could not be inflated owing to perforation.

Remarks. This case is chiefly remarkable for (1) the length of time it lasted, especially after the fluid had become putrid. (2) The absence of any characteristic hydro-thorax breathing. (3) The entire freedom of one lung from pleurisy though the chest-wall was affected. (4) It is nearly but not quite a case of pure pleurisy; there was absolutely no pneumonia in spite of the two abscesses, with the exception of a short narrow strip along the lower border of the left lung. Properly speaking the case should be classed with the pleuro-pneumonia series, but it so narrowly escaped being one of pure pleurisy that I have included it here.

Case IV.

Short History. This horse came in with severe rigors, high temperature, and suspiciously dull chest; the pulse was good and full, and therefore misleading. On the second day pneumonia was undoubted. On the third day the pulse was very small, temperature high, grunting, and patient extremely ill. On the fourth day worse, no appetite, breathing very laboured. Suspect effusion though friction has never been detected, and pleurisy not hitherto suspected. Fifth day, tapped the right chest and got a little blood-stained fluid; tapped the left chest in two places and obtained nothing; towards the evening there was very great distress, sweating, and lying down. Sixth day, intense respiratory distress; tapped both sides and drew off many quarts of blood-stained serum having a distinctly fœtid odour. During the whole day there was great distress, and death occurred in the evening. The heart sounds before death were single.

Pulse, Respirations, and Temperature. On admission the pulse was 56, and it did not rise to 80 until the end of the fourth day; the breathing throughout was rapid. The temperature was 106° on admission, and only once below 105°.

Chest Sounds. First day, dulness of both chests below. Second day, dulness extending up, and even at deep inspiration nothing can be heard. Third day, no air whatever entering lower half of both chests, tubular breathing in left chest. Fourth day, tubular breathing over the entire lower half of both chests, though at this time the cavity contained a considerable amount of fluid.

Outline of Post-mortem Examination. The lungs could not be inflated, but there was a rupture of the pleura in the anterior lobe of the right lung. The pleural sacs did not communicate; the fluid in the right was turbid, that in the left quite clear. There was general pleurisy of both cavities, but the lungs though compressed and carnified as a result were otherwise healthy.

Remarks. (1) It is curious that the advent of pleurisy should be ushered in by symptoms which pointed to the lung and not the pleura. Pleurisy was not suspected until effusion occurred. (2) It is strange no fluid was obtained when the chest was tapped on the fifth day, though an abundance was obtained on the following day. (3) The tubular breathing heard at the bottom of the chest was the sound conveyed through the fluid, there being no lung here whatever. (4) The case is an example of pure pleurisy.

Case V.

Short History. This case was admitted with pleuritic friction of the left chest, and a suspicious dulness of the lower lobe of the right lung. The pulse was 60, and the respirations only 12. On the second day the temperature fell to 101°, the respirations still remaining at 12. On the third day the area of pleuritic friction in the left chest was greater, and there was also an absence of respiratory murmur from the inferior lobe; in the right lung well-marked pneumonia was present, accompanied by tubular breathing; there was also a considerable rise in temperature,the appetite was poor, and an amber-coloured discharge, very common in pneumonia, trickled from the nostril. On the fourth day the temperature was still rising, now reaching 106°, the respirations had gone up to 28, the friction in the left chest had disappeared, while the right chest remained as before. On the seventh day there was an increase in the pneumonia of the left lung and tubular breathing, the right lung had undergone no change, the horse was very ill, no appetite, and as the line of percussion dulness of the right chest was very marked effusion was suspected. Eighth day, respirations were distressed, tubular breathing now heard all over the left chest, and to a greater degree in the right chest; the breathing being more laboured the suspicion of effusion was increased. Ninth day, tapped the left chest, and removed 15 quarts of putrid serum. Thirteenth day, several tappings have been performed on both chests since the last entry, all of these being putrid; great distress in the breathing, and the latter is not characteristic of effusion. Fifteenth day, died early in the morning.

Outline of Post-mortem Examination. There was practically no pneumonia, at any rate no hepatisation; the right lung was compressed to an extraordinary degree, and had suffered from carnification accordingly. In it there was one small abscess about the middle; in the left there were several small abscesses immediately behind the heart, and this lung was only compressed to a trifling extent. In each lung it was the carnified portion which contained the abscesses. There was general pleurisy of both lungs and chest wall, though in the right chest the wall was not affected the whole way up. Both lungs were adherent in several places, and the anterior lobes were firmly adherent to the pericardium. The pericardium was severely affected.

Remarks. (1) Note the complete collapse of the right lung (it was only 1½ inches in thickness at its thickest part), and the very partial collapse of the left. There must have been immense pressure exercised on the mediastina, and further, it is clear that the two pleural cavities did not communicate. (2) The absence of pneumonia and the presence of abscesses in the lung is peculiar; I cannot explain the fact, but it may yet be shown that carnified lung is as suitable a site for necrosis as hepatised lung. On the other hand, as in a previous case of this series, the fact of any abscess being in the lung may preclude it from falling under the head of pure pleurisy. However, I leave it and other cases provisionally under this head.

Case VI.

Short History. This horse was seized with a sharp rigor, and on examining the chest nothing could be made out for three or four hours, when a patch of pleurisy was very evident behind the right elbow; by the evening there was intense friction all over the bottom of the left chest, the pulse was very oppressed, and though the temperature on admission was only 101·6°, it rapidly rose to 105°; pulse 80, respirations 40. Second day, all friction gone; both chests, but especially the left, extremely dull below; temperature fallen and patient better. On the fourth day the temperature fell to normal, pulse 50, respirations 12, and this satisfactory condition lasted until the seventh day, when a relapse took place, temperature, pulse, and respirations rising, but strange to say the chest symptoms were apparently trifling; both sides were resonant on percussion, and there was only slight oppression of the normal murmur. As effusion was suspected, the greatest care was exercised in the examination of the chest. On the ninth day the patient was feeding badly and very ill, the pulse was 80, the respirations 28, but though the effusion is gravely suspected, the percussion note is still clear, in spite of the fact that both lungs give no indication of air passing through them below, and tubular breathing is present behind the right shoulder. Tenth day, the left chest is quite dull on percussion, the right chest resonant, tubular breathing is present in both, intense friction can be heard in the upper third of the left lung, while in both no air can be heard passing through the lower half. The left chest was tapped, and the serum obtained had a slight odour. Eleventh day, tapped the right chest and obtained putrid serum. Twelfth day, died.

Outline of Post-mortem Appearances. The lungs could not be inflated, therefore pneumo-thorax existed. In both lungs a few small abscesses were found, but otherwise they were healthy; no pneumonic tissue surrounded these abscesses, and they were, I think, of pyæmic origin, for between the lungs and diaphragm, on the right side, were three abscesses. Both lungs were much collapsed from pressure and carnified. There was general pleurisy of costal and pulmonary membrane, the entire chest being affected, and also the pericardial sac. Both chests contained a quantity of turbid fluid.

Remarks. (1) This is a somewhat singular case, owing to the chest sounds. I can offer no explanation of the observed phenomena. (2) I have recorded the case as one of nearly pure pleurisy; again exception may be taken to this on account of the abscesses in the lungs, but I think these were secondary, and due to infection from the diaphragm abscesses, for there were none of the ordinary appearances of pneumonia present. On this point the remarks made in previous cases apply. (3) It is curious to observe how commonly relapse follows about the end of a week, especially in those cases which appear to be doing quite satisfactorily; these symptoms are most misleading, and should not throw us off our guard or induce a hasty prognosis.

SOME INQUIRIES CONCERNING THE LATHYRUS SATIVUS.[1]

By JOHN B. WOLSTENHOLME, F.R.C.V.S., F.R.M.S.

IT is noticeable in veterinary and medical literature that this legume is at one time referred to as a "pea," and at another as a "vetch," and then again as "these peas, or vetches, to give them their proper title." This seeming ambiguity has induced me to have some fifty plants reared in a good garden in the country, and to refer to several works and papers on the subject. It is the results of these inquiries I wish to bring forward.

In this country in the corn trade the seeds of the lathyrus sativus are known as "mutters." They vary much in colour, being grey, yellow, light brown, dark brown, or nearly black. They are irregular in shape, being compressed and angular, and vary in size; on the average six placed in apposition measure 1 inch. Lathyrus sativus, the Linnæan name, is the scientific one adopted in France, Holland, and this country. Louden gives chickling vetch as the common British name. Gesse is the French name for the genus lathyrus, and gesse cultivée the name of this particular species (Lamarck). Parkes gives kassaree-dholl as the name by which it is known in India.

Bentham, in his "British Flora," p. 131, uses the term pea as synonymous with lathyrus in his heading of the genus. He also says:—"The pea of our kitchen-gardens and fields is usually distinguished as a genus under the name of pisum, but upon characters which are hardly sufficient for the separation of a solitary species." At p. 127, speaking now of vetches, he says:—"The tendrils distinguish it from all our leguminous plants, except the following genus pea, from which it is absolutely separated, chiefly by the style." With Bentham, therefore, a lathyrus is a pea.

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[1] A communication with specimens to the Lancashire Veterinary Medical Association, 30th September 1897.

Hooker, in his "Flora of the British Isles," divides the sub-order papilionaceæ into six tribes, one of which he names viciæ, which is the Latin equivalent of vetches. This tribe includes all the leguminosæ with tendrils; he then, like Bentham, divides it into the two genera, vicia—vetch, and lathyrus—pea. He ignores a genus pisum, including any so-called with the lathyri.

Bentham describes ten species of vetches and ten species of lathyri, as growing naturally in the British Islands. Hooker describes sixteen of vetches and twelve of lathyri; neither includes the lathyrus sativus.

Lamarck, a French botanist, writing in 1778, divides the leguminous plants with tendrils into two genera—as Hooker and Bentham have done later—using the Linnæan names vicia and lathyrus, with the French equivalents of vesce and gesse. He further says:—"I know no character sufficient to establish a generic distinction between the lathyri and the peas." Lamarck describes thirteen species of vetches and seventeen species of lathyri as growing naturally in France. Amongst the latter is included the lathyrus sativus, a point I wish to draw attention to, as Louden, p. 620, designates it as indigenous to Southern Europe.

Louden in the "Encyclopædia of Plants," divides the leguminous plants with tendrils into three genera, viz.:—Vicia, with thirty-eight species; lathyrus, with thirty species; pisum, with three species.

It will thus be seen that a lathyrus is *not* a vetch, and, further, that, according to Lamarck, Hooker, and Bentham, the term lathyrus includes that small number of species which some detach from the lathyri to constitute the genus pisum. I have quoted from Louden the name of chickling vetch as applying to the lathyrus sativus. It will be necessary to avoid the term vetch, it being most misleading when used in the popular or non-scientific sense, for a number so named do not belong to the genus vicia at all, as—

Chickling vetch	Lathyrus sativus
Bitter vetch	The old genus orobus
Yellow vetchling	Lathyrus Aphaca
Milk vetch	Astragalus glycyphyllus
Kidney vetch	Anthyllus vulneraria.

The following is a description of the plant reared for me from mutters in a rich garden soil:—

Lathyrus sativus, a climbing, branched, glabrous annual, with winged stems, 2 to 3 feet high. Stipules large, lanceolate, half sagittate. Leaf-stalk, flattened and winged, ending in a branched tendril; the stalk bears a pair of long—about 2¼ inches—lanceolate leaflets, three to five nerved. Peduncles axillary, about 2 inches long, bearing a single flower. Flower, standard blue, becoming purple to red as the centre of the flower is approached. Keel pale pink or white. The standard is large, ½ to ⅝ inch in width, rounded, emarginate at the centre, and revolute. The style is flattened below the stigma, quite glabrous on the outer side, but downy on the inner face for a considerable distance below the stigma. The pod is ovate, 1 inch to 1¼ inch long, flattened, and with a double edge at the long axis of the back, *i.e.*, that border to which the seeds are attached. The usual number of seeds is three or four, but I have noticed five. As before described, they are small, varying in size, six measuring 1 inch, compressed and angular. Colour, various—grey, dirty yellow, light brown, dark brown and nearly black.

Louden thus describes the lathyrus sativus, chickling vetch. A deciduous climber, annual. Height 3 feet; flowers June, July; colour light blue. Peduncles one-flowered. Tendrils two and four-leaved. Pods ovate, compressed, with two edges at the back.

Native country, Southern Europe. Introduced into England, 1640.

Lamarck, p. 574, very clearly describes the plant and varieties as gesse cultivée.

Lathyrus sativus, Lin. Sp. 1030.
Lathyrus sativus flore fructuque albo, Tournef, 393.
β Lathyrus sativus flore purpuro, *ibid.*
Lathyrus cicera, Lin. Sp. 1030.

He says that the seeds are eaten as peas.

Lamarck just as clearly describes the common pea as a lathyrus, thus:—

Gesse potagère.
Lathyrus oleraceus.
Pois des jardins.
Pisum sativum, Lin. Sp. 1026.
β Pisum arvense, fructo albo, Tournef, 394.
Pois des champs.

Bouillet's "Dictionnaire des Sciences," etc., p. 762, describes lathyrus as a genus of the papilionaceæ of the tribe vicia. The lathyrus sativus is known by the French names of la gesse cultivée ou domestique, pois breton, pois carré, ou lentille d'espagne. He further states that the seeds are eaten by people as boiled vegetables, and in soup also, and that it furnishes excellent forage for cattle.

I will now give some interesting details culled from Parkes and Louden:—

Parkes' "Practical Hygiene," Edition 1883, page 252, says "In India the use of a vetch lathyrus sativus (kassaree-dholl) with barley or wheat gives rise to a special paralysis of the legs when it exceeds one-twelfth part of the flour; lathyrus cicera has the same effect."

Dr Irvine ("Indian Annals," January 1868) described the symptoms produced by the kassaree-dholl or lathyrus. The first symptoms are gastro-intestinal irritation, and the paraplegia followed on this. Page 256, "lathyrus sativus (kassaree-dholl of India), occasionally in Europe, and constantly in some parts of India, this vetch has been used when mixed with wheat or barley flour for bread. When used in too great quantities it produces (without there being necessarily any alteration of the grain?) constipation, colic, and some form of indigestion, and, if eaten in large quantity, paraplegia. It is also injurious to horses, but less so to oxen. In Bengal Dr Irvine ('Indian Annals,' 1857, *ibid.*, January 1868, page 89) notices the resemblance of the symptoms to the Barbiers of Boutius, and found in some villages no less than 10 to 15 per cent. of the people paralytic from this cause."

In Louden's "Encyclopædia of Plants," p. 620, is the following:—"Lathyrus, a name employed by Theophrastus, to designate a leguminous plant. It is said by his commentator, Bodeus a Stapel, to have been derived from an augumentative principal, and 'θουρος,' anything which is exciting, and 'to have been applied to this plant in consequence of certain aphrodisiacal qualities ascribed to it."

Lathyrus sativus (gesse, French) is frequently sown in Switzerland for *soiling* horses. In several parts of the Continent a white, light, pleasant bread is made from the flour of this pulse, but it produced such dreadful effects in the last century that the use of it was forbidden by an edict of George, Duke of Wurtemburg, in 1671, and this not being observed was enforced by two other edicts under his successor Leopold, in 1705 and 1714. Mixed with wheat flour in half the quantity it makes a very good bread that appears to be harmless. But bread made with this flour only has brought on a most surprising rigidity of the limbs in those who have used it for a continuance, inasmuch that the exterior muscles could not by any means be reduced, or have their natural action restored. *These symptoms usually appeared on a sudden*, without any previous pain, but sometimes they were preceded by a weakness and disagreeable sensation about the knees. Baths, both hot and cold, fomentations, and ointments of various kinds have been tried without effect, inasmuch that it is regarded as incurable, and, being neither very painful nor fatal, those who are seized with it usually submit with patience.

Swine fattened with this meal lost the use of their limbs, but grew very fat lying on the ground. A horse fed some months on the dried herb was said to have his legs perfectly rigid. Kine are reported to grow lean on it, but sheep not to be affected. Pigeons, especially young ones, lose the power of walking by feeding on this seed. Poultry will not readily touch it, but geese eat it without any apparent damage.

In some parts of Switzerland cattle feed on the herb without any harm. It would be worth inquiring, therefore, whether the soil may not contribute something to the ill qualities of the plants, and it is remarked that the seed from a strong, fat, moist soil is much more deleterious than from a light dry one (Duvernoy).

Fabbroni from Florence, in 1786, says that the government here has cautioned the peasants against the use of lathyrus sativus, swine having lost the use of their limbs, and become pitiable monsters by being fed exclusively on this pulse. The peasants, however, eat it boiled or mixed with wheaten flour in the quantity of ¼ without any harm.

The poisonous lathyrus from Barbary is lathyrus semine punctato of casp. bauhin, and seems only to be a variety, for in the crops of lathyrus sativus in Italy they find black seeds striped with white, as in the African seed. Fabbroni suspects it to be a mule between lathyrus sativus and cicera, for the flower and seed partake of the characters of both, having a black seed marked with white, and a white banner with a red keel to the corolla.

I append references of published papers as follows:—

"Cases of Lathyrus Poisoning," by Joseph Leather & Sons—*Veterinarian*, April 1885. Lectures by Principal M'Call: "Poisoning of Horses by the Lathyrus Sativus"—*Veterinarian*, 1886, page 789. "The Bristol Tramways Company's Case"—see *Bristol Mercury*, 5th July 1894. "Laboratory Notes," Professor Bayne—*Veterinarian*, 1894, pages 450–531, and 624. "Vetch Poisoning," by Joseph Abson—*Veterinarian*, 1894, page 754. "Lathyrus Poisoning," by R. F. Wall—*Veterinarian*, 1896, page 587.

EDITORIAL ARTICLES.

THE MODE OF INFECTION IN GLANDERS.

SOME recent contributions to the literature of glanders show that although the main facts of the etiology, morbid anatomy, and prophylaxis of that disease are now well known therestill remain a number of points regarding which there is room for doubt. It is fortunately no longer open to dispute that glanders has but one cause, namely, the transference of the glanders bacillus from diseased to healthy horses, but there is not yet the same unanimity regarding the exact method of infection. It is on all hands admitted that by whatever channel living glanders bacilli gain access to the body of a horse glanders is, or may be, the result; but, while some maintain that the great majority of cases of glanders occurring naturally in the horse are due to the inhalation of bacilli or dust particles containing them, others contend that the ingestion of food or water contaminated with the bacilli is by far the most frequent natural method of infection. Mr Hunting and Professor Nocard are in agreement on this point, both favouring the ingestion theory of infection. The first of these authorities, in a paper which he recently read before the Midland Counties Veterinary Medical Association,[1] deals with this point, but he does not bring to the support of his contention any arguments that are likely to convince those who have hitherto been in doubt regarding this matter. For the most part his case rests on dogmatic assertions based on clinical observation, and for the remainder on a number of assumptions that are either opposed to probability or actually negatived by experience. Mr Hunting says: "The disease spreads from unsuspected horses by the medium of water cisterns, food mangers, pails, etc. Especially in stables using bales instead of stalls is the spread of glanders easy from one horse to another through the food in the manger. There is no protection against an infected horse snorting or sneezing over the food in the next manger." Regarding inhalation as a means of infection, he observes that "it is only a theory founded on analogy. It has no support from clinical observation, and is opposed to all we know of the vitality of the bacillus and the ordinary law of probabilities." This can hardly be regarded as contributing anything new to the question at issue. It is a statement of the opposing views, but it does not supply material from which anyone can form an independent conclusion as to which view is the more probable or the correct one.

The fact is that clinical observation is incapable of resolving this question. It is vain to appeal to cases in which a horse tenanting a stall recently vacated by a glandered horse has contracted the disease. In the first place, those who maintain that inhalation is the common method of infection have never denied that horses may contract the disease by ingestion, and, in the second place, the fact that successive tenants of the same stall have contracted glanders obviously does not exclude the possibility of the animals in question having been infected by inhalation of bacilli voided by previous occupants of the stall. If clinical observation could show that horses introduced into a glanders stable never contract the disease except when they consume food or water containing glanders bacilli the matter would be settled, but no one, unless it be Mr Hunting, has ever pretended that he has any such evidence to put forward. We are therefore compelled to turn in other directions for evidence bearing on the question.

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[1] Veterinary Record, 4th December 1897.

Such evidence is derivable from a study of the common seats of the lesions in glanders, and the relationship of these seats to the channels by which bacilli may readily find entrance to the body. Fortunately, all observers are pretty well agreed as to what are the common seats of disease in glanders. The lungs are incomparably the most frequent seats of glanders lesions. Mr Hunting himself admits this, but he says that "in a few very rare cases we have those so-called 'farcy' lesions without recognisable changes in the lungs." It would have been of interest to know in how many cases this has happened in Mr Hunting's own experience, for we believe it has fallen to the lot of very few, even among those who have had great experience, to meet with a case of pure farcy. That, however, is immaterial from the present point of view. The all-important fact is that in glanders of the horse tubercles are rarely absent from the lungs, and next to that in importance is the fact that in a considerable proportion of cases the lungs are the exclusive seats of the disease. These two facts appear to justify the conclusion that the lungs are generally the primary seats of the lesions. Now, the primary seats of the lesions in bacterial diseases appear to depend upon one of two factors, viz. (1) the anatomical relationship of the part in which the lesions develop to the place at which the bacteria gain access to the body, and (2), certain characters of the part which render it a specially favourable soil for the development of the bacteria. One or two illustrations will make this clear. Anthrax in the pig, and less frequently in the horse, has swelling of the pharyngeal lymphatic glands and surrounding connective tissue for its primary lesion, and this, it is generally agreed, points to infection by bacilli which have penetrated into the tissues from the mouth or throat, and have been carried in the lymph stream to the pharyngeal lymphatic glands. In other words, the glands are here attacked because of their anatomical relationship to the part at which the anthrax bacilli entered the body. Or, to take another illustration, which is perhaps even clearer, the common primary seats of lesions in actinomycosis of the ox are the tongue, the jaw bones, and the lymphatic glands of the throat. Here, again, it is generally admitted that a sufficient explanation of this localisation of the disease is found in the fact that the actinomyces is taken in with the food, and first penetrates into the tissues of the mouth or throat.

Foot-and-mouth disease, on the other hand, furnishes an illustration of the class of diseases in which anatomical considerations do not adequately explain the situation of the lesions. It has erroneously been suggested that the feet and the mouth are the special seats of the lesions in that disease because the virus generally penetrates at these points, but this view is untenable when it is remembered that the fingers and toes are not infrequently the seat of vesicles when the human subject is infected with foot-and-mouth disease by means of milk. To explain the constancy of lesions in connection with the mouth and feet in foot-and-mouth disease one must assume that the virus of the disease, whatever its exact point of penetration, is carried throughout the body, and multiplies with special frequency in the parts mentioned because of something in them which is specially favourable to the settlement or propagation of the germs in them. Another disease of the same class is joint-ill. Here, again, we cannot explain the frequency with which the large joints of the limbs are attacked except by assuming that the bacteria are carried all over the body by the blood, and that the joints are "predilection seats" for their arrest and multiplication.

Let us now see how these considerations apply to the case of glanders. It can hardly be denied that if this were the only evidence available it would have to be declared probable that the lungs are the common seats of lesions in glanders simply because the bacilli are usually carried into these organs with the inspired air. The other view asks us to believe that the bacilli are, as a rule, first taken into the body with food or water, and that pulmonary tubercles are the most constant lesions because the lungs represent predilection seats for the arrest or multiplication of the bacilli. There are several difficulties in the way of accepting this explanation of the facts. If glanders bacilli travel from the intestine or stomach to the lungs it must be either by way of the blood stream or the lymph stream. In either case it is a long journey, involving a passage through the intestinal mucous membrane, and after that through the liver (if by the blood stream), or through one or more lymphatic glands (if by the lymph stream). Now, what one would naturally expect in such a case would be to find glanders lesions developed somewhere on the track of the bacilli, most probably in the mesenteric glands if the bacilli were carried by the lymph stream, or in the liver if they were carried by the blood. As a matter of fact, glanders lesions have been met with in these situations, but it cannot be held that they are at all common, and Mr Hunting even denies their occurrence, and thus effectually deprives himself of an appeal to them in support of the ingestion theory of infection. This is undoubtedly the greatest obstacle to the acceptance of that theory, for it is altogether opposed to observation and experiment to ask us to believe that in nearly every case of natural glanders in the horse the bacilli, without exciting any lesion, pass through the lymphatic glands which guard the entrance to the thoracic duct, or through the liver, which stands between the portal vein and the systemic circulation. Mr Hunting himself asserts that when the bacillus "enters the body through the abraded skin or mucous membrane it causes lesions of a well-known character—nodules or 'buds,' swelling of lymphatic vessels and glands, suppuration and ulceration." No one who believes this and denies the occurrence of abdominal lesions ought to be found supporting the ingestion theory.

Those who hold the exact method of infection doubtful, or who regard the ingestion theory as the less probable of the two, have never denied that the inhalation theory has its weak points. The chief of these is the comparatively feeble vitality of the glanders bacillus in the outer world, and in particular the fact that it is destroyed by complete desiccation. But while it is quite true that complete desiccation of artificial cultures renders the bacilli inert, it has not been conclusively shown that animal discharges may not be so far dried as to admit of their being pulverised without killing the bacilli contained in them. Furthermore, it may be pointed out that the feeble resistance which the glanders bacillus offers to destructive agencies is a fact as little in favour of the one theory as of the other, for one would expect that in the case of an organism so easily killed the gastric juice would be a serious obstacle to infection by way of the intestine.

Mr Hunting makes some curious statements with regard to the mode of dissemination of glanders bacilli throughout the body. He says: "When living bacilli escape from the lungs and lodge in other parts of the body they cause local trouble, but these local changes soon pass through their different phases and disappear, leaving hardly a trace of their occurrence." If glanders bacilli escape from the lungs it must either be by way of the air passages, the blood stream, or the lymph stream. In any of these cases it is difficult to understand the grounds for supposing that they will prove so harmless to the individual harbouring them, in view of the fact that such escaped bacilli are universally held to be the source of infection of other horses. Perhaps, however, the passage we have quoted was put forward merely as an interesting speculation, but in that case it was a pity to present it in the guise of a fact.

As previously observed, M. Nocard is also a supporter of the ingestion theory of equine glanders, and his views on that point are very clearly stated in the article which appears in this number. He maintains that the only means by which one can experimentally obtain miliary tubercles in the lungs identical with those of natural glanders is to cause a horse to ingest materials containing virulent glanders bacilli; and he feels driven to conclude that ingestion is the common method by which glanders is naturally contracted. In fact, M. Nocard appears to be logically bound to draw a wider conclusion than this, for he would evidently ascribe all cases of glanders with pulmonary tubercles to alimentary infection, and practically that includes all the cases of natural glanders that are met with—at least in this country. It is important to observe that he admits that glanders may be experimentally communicated by various other methods besides ingestion, but contends that when lung lesions follow cutaneous, subcutaneous, intravenous, or intratracheal inoculation, they assume forms that are quite different from the common glanders tubercle met with in natural cases of glanders.

The question may now be asked, Do Professor Nocard's experiments completely justify even the more restricted conclusion, viz., that ingestion is the common method of natural infection in glanders? We think this question ought to receive a negative answer. In the first place, M. Nocard surely makes too sweeping a statement when he affirms that the common glanders tubercle of the lung cannot be experimentally produced by any other method of infection than ingestion. It must, of course, be admitted that M. Nocard's own experiments prove that horses can with great certainty be infected by feeding with materials containing virulent glanders bacilli, and that this method of infection determines the formation of pulmonary tubercles identical in appearance with those found in natural cases of the disease. So far as we are aware, no one has ever denied either of these statements, and the admission of them is not at all inconsistent with the view that the majority of pulmonary glanders tubercles are due to the propagation of bacilli that entered the lung with the inspired air. Professor Nocard cannot know as a matter of certainty that glanders bacilli introduced by other channels than the alimentary canal never produce pulmonary tubercles of the ordinary character, but he may be in possession of a certain amount of experimental evidence pointing in that direction. If he is, it is matter for regret that the evidence has not been published. We are told that when glanders follows infection through a wound of the skin or mucous membrane, the resulting lung lesions are of the nature of more or less extensive infarcts, or of areas of pneumonia or broncho-pneumonia similar to those met with in experimental glanders of the ass, but never ordinary tubercles. Experiments on an extensive scale may in the future compel us to accept that statement as correct; but in the meantime it is legitimate to urge against it that it is inherently improbable. If a horse is inoculated, say, on the hind leg, and from the primary lesion there bacilli are subsequently transported to the lungs, such transport must be effected either by way of the blood-stream or the lymph streams. But these are also the channels by which bacilli must reach the lung from the intestine, and yet we are asked to believe that with great constancy the bacilli that reach the lung from the intestine lead to the development of miliary tubercles, while those that come by the same vessels from a cutaneous or intravenous inoculation provoke the formation of an entirely different type of lesion!

Our readers can hardly fail to notice that in M. Nocard's article there is a lack of information regarding a point that is all-important, to wit, the condition of the abdominal organs in the animals experimentally infected by ingestion. It is very expressly stated that typical glanders tubercles were found in the lungs of the experimental horses, but it is not said whether the intestines, the mesenteric glands, and the liver, in which one would naturally expect to find lesions, were healthy or not. If these parts were all free from lesions the fact must be accepted, remarkable though it is, and in that case it would also have to be admitted that one of the main objections to the ingestion theory had been removed. On the other hand, if in any large proportion of the experimental horses glanderous lesions were present in the intestine, liver, or abdominal lymphatic glands, then the experiments would be opposed to the ingestion theory, in view of the fact that abdominal lesions are admittedly exceptional in natural cases of glanders.

It may be thought by some that the amount of space devoted to the discussion of the mode of infection in glanders is out of all proportion to the importance of the subject; but, in opposition to that view, it may be pointed out that in this as in other diseases it is of considerable practical interest to know what is the exact manner of infection, for such knowledge can hardly fail to give better direction to our efforts to prevent the spread of the disease.

It cannot be denied, however, that the most interesting part of M. Nocard's recent researches is that which bears upon the natural course of glanders in the horse. A few years ago the orthodox view of the disease, and the only one that was justified by the then available evidence, was that glanders, in the great majority of cases, entailed death within a comparatively short period—sometimes only a few weeks, and seldom more than a few months. Since the introduction of mallein it has become clear that this was an entirely exaggerated estimate of the gravity of the disease, for, contrary to expectation, it has been found that many animals proved by the mallein test to be glandered survive for periods of months or years afterwards in apparent health. M. Nocard's recent experiments carry us a point further than that, for they prove that a very considerable proportion of glandered horses make a perfect recovery from the disease. A conclusion so utterly opposed to common experience as this appears to be is not likely to find immediate general acceptance, but, as a matter of fact, the results of M. Nocard's experiments do not conflict with clinical observation.

Prior to the introduction of mallein clinical observation had led to the conclusion that glanders was incurable, but what it really proved was that, with few exceptions, horses that develop external symptoms of glanders soon succumb to the disease, and that is still the common experience. But now, thanks to mallein, we know that in most outbreaks of glanders the number of horses that at any given moment show external symptoms bears a very small proportion to those that are actually diseased. A good many veterinary surgeons in this country have had the opportunity to observe that of the apparently healthy horses found to be glandered by the mallein test many continue in apparent health for periods far beyond what used to be reckoned as the incubation period of glanders, but the circumstances have seldom or never furnished the data from which one could determine whether the disease merely continued to be "latent," or whether actual recovery had taken place. This is the point that M. Nocard's experiments have cleared up. The proof of recovery does not rest merely on the cessation of reaction to mallein, which might reasonably have been regarded as perhaps a temporary effect (like that which often follows a first injection with tuberculin) and not inconsistent with the continued presence of living glanders bacilli in the body of the animal. The proof rests on the fact (1) that the lesions found in horses that had ceased to react to mallein were non-infective when inoculated into such sensitive animals as guinea-pigs or donkeys; (2) that it was impossible to cultivate glanders bacilli from these lesions in artificial media; and (3) that horses that had ceased to react to mallein did not convey the disease to their stable companions.

The conclusive demonstration of the fact that many glandered horses not only remain free from every external symptom of the disease for a long period, but also actually recover from it, has a most important bearing on the prophylaxis of glanders.

We learn from M. Nocard that the method of dealing with outbreaks that is generally followed in France comprises the destruction of every horse showing external symptoms of glanders, the employment of the mallein test for all the horses of the infected stud, the separation of those thus found to be healthy from those already affected, and the periodic testing of the latter, to be followed by their slaughter when they develop clinical signs of the disease, or by their return to the healthy moiety of the stud in the event of their ceasing to react to mallein. M. Nocard tells us that although this plan is not enforced by law in France it is generally voluntarily put into action by the owners of diseased studs, which would appear to indicate either that the French horse-owners are more intelligent than the members of the same class in this country, or that the former are the recipients of better advice from their veterinary attendants.

London continues to be the great hot-bed of glanders in Great Britain, and the owners of glandered studs in the metropolis, or at least the great majority of them, have not yet awakened to the fact that they might with comparatively little trouble and expense get rid of the disease. In a few studs mallein has been employed to effect a permanent separation between the diseased and the healthy, but for the most part it is used simply as a final test in cases presenting clinical signs of glanders. The consequence is that very little progress in stamping out the disease is being made, and for this unsatisfactory state of affairs some members of the veterinary profession in London are in no small measure to blame. The owners of glandered studs are told that they are worthy objects of public commiseration, that it is their misfortune and not their fault that the disease exists in their stables, that they play the part of public benefactors when they kill their clinically glandered horses, and that Local Authorities are guilty of meanness and ignorance when they refuse liberal compensation for the distruction of such animals. To the public it is represented that the great obstacle to the successful operation of the glanders Order of 1894 is the withholding of reasonable compensation for the horses slaughtered as glandered, and that as long as compensation is inadequate owners will try to evade the law and refuse to notify the existence of the disease. The effect of this pernicious teaching is that the majority of the owners of glandered studs refrain from putting into action those measures which would, to their own great profit, stop the further spread of the disease among their animals.

It is no doubt a misfortune for a man to become the possessor of a clinically glandered horse, but it is not an entirely unpreventable misfortune. Examination by a competent veterinary surgeon prior to purchase is a tolerably certain safeguard against such a calamity, and where it fails the loss can be thrown on the veterinary surgeon who made the error. Of course it must be admitted that this affords no guarantee against the purchase of a horse actually glandered though apparently healthy, but neither would compensation for the slaughter of clinically glandered animals ward off this danger. There is, however, a measure that would soon reduce this risk to the vanishing point, viz., to treat every animal found in a glandered stable as suspected of glanders, and prohibit its sale until it has been proved free from the disease by the mallein test. We are not recommending such a plan, but merely pointing out that in that direction, rather than in liberal compensation for visibly glandered horses, lies the remedy for the present dangers.

But while there is perhaps an element of misfortune in the purchase of a glandered horse, it would in the present state of our knowledge be a misapplication of the word to describe the continued existence of glanders in a stud as a misfortune.

The continued existence of glanders in a stud of horses is a state of affairs for which the most indigent owner has at the present time no excuse. His stock of horses may comprise a few that are clinically glandered, and a larger number that have the disease in the latent form, but, unless there has been the grossest negligence the great majority of the animals will be healthy. We are given to understand that because only carcase value is given for the animals visibly affected, the owner is compelled by motives of economy to conceal the existence of the disease as long as possible. But has he not a veterinary surgeon to point out to him the madness of such a course, and to advise him that there can be no economy in keeping in his stock such a fertile source of contagion as a horse showing clinical signs of the disease? Has he not been told that if he will kill the visibly diseased animals, test the remainder with mallein, isolate those that react, and practise thorough disinfection, he may fairly calculate upon getting rid of the disease at a cost which will be trifling compared with his own plan of temporising with it in the vain hope that the Local Authority is going to compensate him for the visibly glandered animals, which have caught the disease mainly through his own negligence?

There is also an element of truth in the argument that liberal compensation for visibly glandered horses would lead to the early notification of a good many cases that are at present concealed as long as possible, but it is hardly to be credited that quarter value, which we are told would satisfy the London horse-owners, would yield any such result. If full value were given, then, no doubt, the owner would promptly notify symptoms of glanders, for the money paid him in compensation would buy him a healthy horse to take the place of the one destroyed. But, one fails to see in compensation to one quarter of the animal's value any inducement to early reporting that is not at present in existence. The unscrupulous owner would still be tempted to work the horse as long as possible, in the knowledge that whether he notified the existence of the disease early or late he would be awarded compensation. Indeed, everyone who desires to see glanders stamped out of London must object to more than carcase value being given in compensation for horses actually diseased, on the ground that more liberal compensation would actually remove one of the main incentives that owners of glandered studs at present have to try to get rid of the disease.

In the large London studs in which glanders has obtained a footing the annual loss which it entails is represented almost entirely by the slaughter of such animals as develop clinical signs of the disease. The slaughter of all these animals represents a dead loss, but owners know, or their veterinary surgeons know, that the longer clinically glandered animals are kept in the stud the larger is likely to be the proportion of animals that will afterwards develop symptoms of the disease, and have to be killed. In short, the penalty which the owner pays for neglect of the disease is the compulsory slaughter of the visibly glandered animals without compensation, and the penalty for concealment of the disease is a larger crop of cases of glanders afterwards. To give liberal compensation would be to mitigate the penalty, with the almost certain result that the owner would be even more negligent than he has been in the past.

The most fallacious of all the arguments in favour of liberal compensation for the slaughter of clinically glandered horses is the one which represents that such compensation would ensure early notification, and thus put an end to the spread of the disease. Such an optimistic view is absolutely unwarranted. It implies that glanders is spread, either entirely or mainly, by the traffic in horses that are clinically glandered, and that is certainly not the case. So far is it from being the case that if every horse with external symptoms of glanders, on, say, the 1st of January 1898, could in a moment be wiped out of existence, we should still be almost as far off the extermination of the disease as before. Glanders at the present time is mainly spread from stable to stable not by the traffic in clinically glandered horses, but by the sale and purchase of horses that have the disease is the occult form, and no plan which fails to take note of this fact is deserving of serious consideration.

The endeavour to make it appear that, in respect of the equity of granting compensation, glanders is on all fours with cattle plague and pleuro-pneumonia is plausible, but it requires us to overlook a very crucial difference. It is true that in the case of these cattle diseases liberal compensation was given for the diseased animals, but the Local Authority or the Board of Agriculture did not squander money by compensating for the visibly diseased while allowing the in-contact animals to go free, which is what the London horse-owners and some of their veterinary advisers are anxious to persuade the London County Council to do in the case of glanders.

This is really the crux of the compensation question. The London horse-owners have not yet given any indication that they are willing or anxious to have glanders attacked by the only method that is capable of stamping it out. They clamour for compensation, but by that they apparently mean compensation for horses that are likely to die from glanders, or are so manifestly diseased that they dare not work them on the streets. They do not wish any notice to be taken of their apparently healthy but in reality glandered horses, and we believe that we are not misrepresenting them when we say that they would be up in arms at the bare proposal to test every in-contact animal by mallein, and impose on the movement of such animals as react the restrictions that are necessary to put a stop to the disease. As long as that is their attitude we trust that the Local Authority will persist in its refusal to give more than carcase value for horses slaughtered on account of clinical symptoms of glanders. On the other hand, we hope that when the horse-owners declare themselves ready to have every in-contact horse tested with mallein, and all horses that react relegated to special stables, the Local Authority will grant liberal compensation for clinically glandered animals.

THE DISCUSSION ON PLEURO-PNEUMONIA AT THE MONTREAL MEETING OF THE BRITISH MEDICAL ASSOCIATION.

THE question whether contagious pleuro-pneumonia has or has not been stamped out of the American Continent is not one in which the people of this country can now take a great interest, since the importation of cattle from abroad, except for the purpose of slaughter at the port of landing, has been put a stop to by Act of Parliament. It will be remembered that even before the passing of that Act the introduction of store cattle from Canada and the United States of America was prohibited by the Board of Agriculture, because the Board was not satisfied that either of these countries was free from contagious pleuro-pneumonia. Strenuous efforts were made by the Canadian Government to obtain a free entry for its store cattle, and to persuade the Board of Agriculture that the Canadian authorities had no knowledge of the existence of the disease in question in any part of Canada, and that it was therefore reasonable to conclude that the disease did not exist there. To representations of that sort the Board of Agriculture had to reply that among Canadian cattle killed at the port of landing in Great Britain, the British veterinary inspectors from time to time encountered lung lesions which they were unable to distinguish from those of contagious pleuro-pneumonia. We have no intention to discuss at length the evidence bearing on the points in dispute between the Board of Agriculture and the Canadian Government, and we are only induced to refer to the subject here by observing that an attempt was made to revivify the controversy at the meeting of the British Medical Association at Montreal in September last. A discussion on the subject was opened by Professor Adami, and we desire to offer a protest against the form of that gentleman's argument. He is reported to have said that with one exception the authorities who supported the attitude of the British Board of Agriculture on the pleuro-pneumonia controversy had not done any first-class work. Professor Adami, it is perhaps needless to remark, is himself one of those who supported the view of the Canadian Government, and we may infer that he holds rather a high opinion of the merits of his own scientific work. If he maintains that it has all been of the first class far be it from us to contradict him. We would, however, suggest that his opinion on that matter may be biassed, and that in any case it is conceivable that he may hold wrong opinions regarding the pleuro-pneumonia question. Moreover, it is possible that those who differ from him on this point may be right although they have never done any first-class work. In short, Professor Adami set a bad precedent for scientific discussions when he resorted to disparagement of his opponents, and it may be charitably concluded that he spoke without notes, and did not expect his speech to be reported, at least on this side of the Atlantic.

Reviews.

Exercises in Equine Surgery. By P. J. Cadiot, Professor at the Alfort Veterinary School. Translated by A. W. Bitting, D.V.M. Edited by A. Liautard, M.D., V.M. With 56 Illustrations. New York: W. R. Jenkins, 851 Sixth Avenue.

They manage these things better in France. At the Veterinary College, Alfort, facilities are afforded the students to acquire skill and dexterity in operating. Throughout the Session a day every week is devoted to operations conducted mainly on the dead subject. To assist the students Prof. Cadiot issued in 1895 an excellent guide to various operations, many common, some rare, and all more or less useful. The directions are simple and precise, and the illustrations form not the least attractive feature of this very valuable book.

To English readers good translations of German or French veterinary works are always welcome, but, unfortunately, the capacity necessary to the making of an acceptable translation does not belong to everyone who attempts the task. Cadiot's "*Exercices de Chirurgie Hippique*" has been translated by Prof. Bitting, and published in New York. According to the Editor, Professor Liautard, whose "additions and corrections," it must be stated, are not very obvious, the translation was prepared as a graduation thesis at the Iowa Veterinary College, for which praiseworthy purpose it was probably good enough, but as a manual for students and practitioners, read by the light furnished by the original, this reproduction cannot be regarded as an unqualified success. The drawings have been faithfully reproduced, but the text is too literal—indeed, in places hardly intelligible. As Mark Twain remarks of a different book, "no translation could be closer . . . it is as succinct as an invoice."

With the operations it is unnecessary to deal, as the translator is not responsible for the surgical methods of the author; otherwise he has much to answer for. He has produced a translation which has been spoiled to some extent by loose diction, faulty collocation, some errors in spelling, and numerous Americanisms—which may or may not be justifiable. Space forbids detailed reference to the many defects of this irksome book, and the few which follow should be taken merely as samples of the less objectionable. "Pass the lead strap . . ." (pp. 3-8). ". . . When the vessel is opened and in condition so that the blood flows" (p. 8). "Section the tegumentary membrane and the arch of the vascular circumflex . . . " (p. 11). "Make a deep incision of a half a centimeter . . . " (p. 12). "Make two small incisions through the hair" (p. 15). "Place yourself in profile near the limb, back turned toward the rear" (p. 16). ". . . Pass them . . . over the lean flesh" (p. 17). ". . . Detach the skin from a circular surface, within the incision from the inferior radius" (p. 19). "Pass all the threads first, afterwards commence by tying those in the middle. . . . In tieing (sic) the threads . . ." (p. 23). "The lips are brought face to face" (p. 24). Bayer's suture . . . is convenient for those wounds with uncovering of the skin" (p. 26). "If two limbs are to be operated upon, cauterise the internal face of one, and the external face of the other. If two limbs are cauterised, operate upon the internal face of one, and the external face of the other" (p. 26). "One gradually augments the duration of the application of the cautery to the same degree as it cools" (p. 29). "With a second cut of the bistoury separate the sternohyoidean and sternothyroidean muscles upon the median line; these muscles and the cutaneous lips should be divided by means of tenaculums . . ." (p. 35). "In passing the catheter . . . take a position even with the right flank" (p. 48). "The operatory act essential to urethrotomy . . ." (p. 49). "The thread passes through the three intestinal walls; the ends are curved" (p. 73). "Suture for abrasion; muco-mucous suture by infection" (sic) (p. 76). "On the internal face of the hough" (p. 96). "During the execution of this last manoeuver (sic) . . ." (p. 108). The directions for bandaging a foot after operation for sandcrack suggest dancing at a wake. "Cover the wound with wadding of turf . . . make circles around the phalanges and reverse upon the plantar region. Envelope the dressing in a sheet of linen and furnish the whole with a double plait of straw." Again, "Cover the wound with phlegdets (sic) superposed to overflowing over the sides and above the gap. Secure them with a roller bandage; passing the first, turn to the middle, the second above the third, and the balance successively above and below, making as many reverses as may be necessary, being careful that each turn of the bandage covers the inferior two-thirds of that which preceded, and being covered in its inferior two-thirds by the turn that follows . . . All turns of the bandage should be passed behind between the end and the branches of the shoe" (pp. 108-9). It may be that these dressings are first-rate and even easy to apply, but the description conceals their advantages. Directions, whether original or translated, should be so worded as to be easily understood. Puzzles in bandaging and minor surgery only serve to evoke expressions which few persons care to write. Prof. Bitting's book has some good points, but these are neither sufficiently striking nor important to merit special mention. It would be advisable to have the proofs of the next edition carefully revised by someone conversant with practical equine surgery. J. M.

Outlines of Veterinary Anatomy by O. CHARNOCK BRADLEY, M.R.C.V.S., Professor of Anatomy in the New Veterinary College, Edinburgh. London: Baillière, Tindall & Cox.

TWELVE months ago we reviewed the first part of this work, and during the summer the remainder of the book has come to hand. A careful perusal of Parts II. and III. has not changed the opinion we expressed upon Part I.—indeed we are more than ever convinced that book-making has been the object, and not a serious effort to give us an original work. We cannot understand what method, if any, the author has followed; we have not here a systematic work, nor can it be of much value as a dissecting guide. That it may assist a student who already knows his work and has a short time at his disposal for rapid revision we do not doubt, but then he will not fail to find mistakes, some trivial, others serious, several amusing if not ludicrous.

Moreover, we must offer our opinion that if "the actual organs of the ox, sheep, pig, and dog do not commonly find their way into the dissecting rooms" (preface, Part II.), then the veterinary student, if he has to trust to text-books for his knowledge of comparative splanchnology, is in a very poor way indeed. Nor do we think that the illustrations in the present work will help him, since, for example, the spleen of the sheep is represented on the same page as about three times the size of the same organ in the cow!

Exigencies of space forbid us from giving more than a few of the errors, but we may briefly note in Part II. that the mediastinum is not a space (45); that the thymus gland is to be found (a fact that is not mentioned) between the layers of the anterior mediastinum during early life; that the bronchi have not a separate innervation from the lungs (48-49); that the foramen ovale is not present in the adult heart, though the fossa ovalis may be less distinct in the left auricle than in the right (53); that the endocardium itself has no colour; that the os cordis does not arise from ossification of the auriculo-ventricular rings (54); that (79) the dura mater has not dilated and contracted portions agreeing with those of the neural canal. It responds to the cord and not to the neural canal. The author confuses outlet with inlet of the pelvis, and less than six lines is considered sufficient for the description of the peritoneum. Brünners glands are misplaced; they are submucous in position (86). The structure of the mucous membrane of the abomassum is said "to be similar to that of the stomach of the horse" (148). The longitudinal fibres of the rectum that have insertion into the anterior coccygeal bones are erroneously described as forming the levator ani muscle. Retractor ani and levator ani are synonyms. We are glad to see that the correct nomenclature is applied to the lobes of the liver. The apology for venerable authorities at the foot of page 93 was wholly unnecessary. It is unusual to describe the kidney as possessing a serous covering (102). The ligaments of the bladder are unnecessarily multiplied, and the anterior false ligament is described as containing the urachus, "which is the remains of a fœtal structure." The urachus was an important fœtal duct, and, of course, lost its vocation when intrauterine life ended. The scar found at the fundus of the bladder is the indication of the attachment of that fœtal duct (108). The cremaster muscle forms no part of the covering of the testicle; it is a dependency of the obliquus abdominis internus muscle, and a constituent of the cord (115). Cowper's glands has a structure of its own, and does not resemble that of the prostate gland.

On page 116 appears the following: 2 "*Erector Penis.* This is a small muscle passing between the tuber ischii to the penis, raising and retracting that organ." This is very funny, or would be if the author had been writing a burlesque, but it would be a serious blunder for a student to make if he gave the above description in an examination. It savours strongly of Strangeways. Chauveau would have been more trustworthy.

As to the comparative portion in this part, it is a mere abridgement of the larger works, and there are certain important omissions. The commonest papillæ of the rumen, the foliaceous, are not mentioned. The large intestine of the pig is said to be similar to that of the ruminant, but whether in appearance or disposition within the abdominal cavity is not stated. The most striking feature of the pig's liver, its pronounced lobulation, is not even noticed (152). "Two glandular diverticuli open *out of the anus* of the dog" (sic); the anus itself is usually defined as an opening. Cowper's glands are said to be absent from the pig. Gärtner's canals in the cow are said to "pass from the lateral walls (of the vagina) to near the meatus urinarius," and on the same page "*the vulva* of the pig has Gärtner's canals as in the cow."

In Part III., which to our mind is the best of the three, there are certain errors which should not have appeared. A typical vertebra is described (55) and figured (54) as possessing a vertebral foramen; this it does not any more than its transverse process possesses one or more articular facets. The foramen cæcum of Morgagni is described as found in the tongue of the horse. It certainly is not. Chauveau wrongly describes and figures a circumvallute papillæ as the foramen cæcum (2nd English edition, 403). The foramen cæcum as observed in the human tongue is a blind pit placed behind the intersection of the converging lines upon which the circumvallate papillæ are found. It is said to represent the fœtal thyro-glossal duct. Comparing pages 97, 106, 107, and 108, we find that the molar glands have escaped description. On 114 the author confuses Stenson's canal and Jacobson's organ. The canal that has an opening upon the floor of the nose, and in the horse terminates blindly in the cartilage of the naso-palatine cleft, is the canal of Stenson. Jacobson's organ passes backwards from Stenson's canal up the nose and lies embedded in the connective tissues close to the attachment of the septum nasi to the vomer. It has a cartilaginous framework, is some 4 or 5 inches long, and is accessory to the sense of smell. On 116 the foramen in the wing of the thyroid cartilage is ignored, and on 121 the thyro-arytenoid muscle is said to "govern the tension of the vocal cords;" if this means to relax the cords it is correct. The portion dealing with the brain is the best in the whole work; it is very concise and so far as it goes we have no fault to find, though we might here and there think it did not go far enough. But we have said sufficient; we have endeavoured to read the book as the author desires us to do in his preface, and the observations we have made have been made in no carping spirit. Descriptions are right or wrong, inadequate and thereby often misleading, or verbose and frequently muddlesome, and though we do not wish to say that any of these apply to the work as a whole, yet we think in many places more might have been given with profit to the reader. In the event of a second edition we hope for more from the author himself than a mere arrangement of the subject, and trust that the many signs of haste which we have pointed out will be rectified. A. E. M.

Diseases of the Dog and their Treatment. By Dr Georg Müller, Director of the Clinic for small animals at the Veterinary High School at Dresden. Translated, revised, and augmented by Alexander Glass, A.M., V.S., Lecturer on Canine Pathology at the University of Pennsylvania, with ninety-three illustrations. Philadelphia: W. Horace Hoskins, 1897.

It was once observed of the biography of a great man recently deceased that the author of it had added a new terror to death. With equal cause it might be said that the translator of this work has added a new terror to the lives of German veterinary authors. The original work is a book of considerable merit, and it well deserved to be made accessible to the English-speaking veterinary student. We greatly regret that the translator has performed his task so atrociously as to make it impossible for a conscientious reviewer to say a favourable word of the result of his labours. Nearly every page of the book, including even the author's preface, contains passages that are mangled almost beyond recognition, and the translation is bad, not only in that it frequently gives an erroneous rendering of the author's meaning, but also because the product is execrable English. Hundreds of passages might be quoted to show that the Mr Glass is not familiar with German, and, what is much more discreditable, is unable to arrange the words of his own language grammatically. Bad as this is, it does not make up the sum of his offences, for he omits important statements found in the original, and by way of compensation puts into the mouth of the German author expressions that he never used. Occasionally Mr Glass appears to have had recourse to a German-English dictionary to help him out of a difficulty, but in a good many cases he seems to have adopted the easier course of simply omitting the troublesome word or sentence.

Having made this formidable indictment it is only fair to the translator that we should cite a few passages to enable the reader to judge for himself. Here are a few samples:—

In the author's preface we find the following curious passage:—"The author has endeavoured to establish the symptoms with their relation to the disease, and to confine their therapeutic treatment to a knowledge of normal and pathological anatomy and physiology, for he believes that it is on a clear and accurate knowledge of the normal and pathological structure of life the fundamental base of all clinical science lies." It is hardly necessary to assure the reader that the author never said anything so absurd. The passage in the original is—"suchte er . . . auch durch stetigen Hinweis auf die Lehren der normalen und pathologischen Anatomie und Physiologie das Zustandekommen und den Zusammenhang der einzelnen Krankheitsymptome klarzulegen und die zu treffenden therapeutischen Massnahmen zu begründen. Es wird niemand leugnen können, dass die Kenntnis des normalen und pathologischen Baues und Lebens die Grundlage der gesamten Klinischen Wissenschaften darstellt." Which being translated means—"and he has also endeavoured, by constant reference to the teaching of normal and pathological anatomy and physiology, to explain the mode of origin and the connection of the particular symptoms of disease, and to find a ground for the appropriate therapeutic measures. No one can deny that a knowledge of normal and pathological structure and function forms the basis of the whole of the clinical sciences."

The article on tuberculosis is introduced by the following sentence:—"Under this name we class all affections which owe their origin to a peculiar specific bacteria (sic), known as tubercle bacilli." A little further on we are informed that the bacilli appear under the microscope "in the shape of very thin tube-like casts, showing a certain activity of movement." The German of which this is a translation reads:—"sehr dünne Stäbchen, welche keine Eigenbewegung besitzen," that is to say, "very slender little rods which do not possess the power of movement."

One other sample must suffice. Ehrlich's method of staining the tubercle bacillus is given thus in the translation:—"Ehrlich advises that the observer place the secreted matter in a very thin layer upon a covered plate, and allow it to dry in the open air. When this is done pass the glass three times slowly over the flame of a gas or alcohol lamp. Then place it in a watch-glass which contains a coloured solution of sputa." The following are the sentences of which the foregoing is supposed to be a translation:—"Man trägt (nach Ehrlich) den zu untersuchenden Auswurf in möglichst dünner Schicht auf ein Deckgläschen auf und lässt ihn (an der Luft) eintrocknen. Nachher zieht man das Deckgläschen dreimal langsam durch eine Gas- oder Spiritusflamme und legt es dann, mit der Sputumseite nach unten, in ein Uhrschälchen, welches eine bestimmte Farbstofflösung enthält." When correctly rendered this passage reads:—"The sputum to be examined should (according to Ehrlich) be spread in as thin a layer as possible on a cover-glass, and allowed to dry (in the air). After that the cover-glass is drawn three times slowly through a gas or spirit flame, and then laid, with the sputum side downwards, in a watch-glass containing a definite staining solution."

Examples such as these, which appear to be the product of combined ignorance and carelessness, occur with such frequency that the reader who has not the original work beside him could never feel safe in accepting any single statement as emanating from the German author. The best thing that the translator could do, for the sake of his own reputation, and for the credit of the school to which he is attached, would be to destroy the entire edition, and abstain from further efforts in the same line until he has become fairly familiar with the German language, and has found a friend whom he can trust to revise his English. J. M'F.

The Annual Statistical and General Report of the Army Veterinary Department for the Year ending 31st March 1897

THIS Report, like its recent predecessors, records a very satisfactory condition of the Army with respect to the health of its horses. These comprised, on the 31st March 1897, 13,386 troop horses, 109 mules, and 1796 chargers. The amount of inefficiency from diseases and injuries was 61·10 per cent., or 1·09 per cent. higher than during the previous year.

The number of deaths from all causes was 332, or 2·52 per cent. of the average strength. This was thirty-six deaths, or ·30 per cent., more than last year. Of the deaths 1·48 per cent. died, and 1·04 per cent. were destroyed. The greatest mortality was in the Infantry Transport, and next in order came the Heavy Cavalry, the Army Service Corps (which has the Remount Establishment attached to it), the Light Cavalry, the Horse Artillery, the Medium Cavalry, the Field Artillery, the Royal Engineers, the Household Cavalry, the Military Mounted Police, and the Mules.

Exclusive of 284 remaining on the 31st March 1896, the total number of admissions to treatment was 8068, or 61·10 per cent. of the average strength, this being seventy-three more than in the previous year. The average number of days each horse remained under treatment was twenty-two, being one day more than in each of the four previous years. There was a decrease in the number of admissions for surgical diseases and injuries and specific diseases, and an increase under the heads of general diseases, and diseases of the chest and air-passages. The Director-General has again to record the very satisfactory fact that no cases of glanders or farcy had occurred among the Army horses during the year. The Report also gives statistics regarding the work of the Army Vaccine Institute, which show that under Veterinary-Major Longhurst and Veterinary-Captain Butler that institution in an unostentatious way continues to render very great service to the Army.

CLINICAL ARTICLES.

LAPAROTOMY IN A HORSE.

By FRED. HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

THE subject was a brown cart mare about nine years old, a patient in the practice of Mr John Bell, M.R.C.V.S., of Catford. It is to this gentleman that I am indebted for all the clinical history of the case, and for assistance during the operation. To Mr Browning, M.R.C.V.S., College Tutor, I am indebted for the administration of the anæsthetic.

On the morning of the 20th November the animal was taken with colic, the most noticeable symptoms being fits of pain at short intervals and a peculiar manner of kneeling down, with the hind quarters elevated as much as possible. No fæces had been passed during the previous night, and the animal refused all food. Temperature was normal, the pulse and respirations slightly accelerated. An antispasmodic draught, containing extract of belladonna, oil of turpentine, and linseed oil, failed to give any relief.

At night a purgative dose of aloes was administered. Examination per rectum revealed that organ to be almost empty, whilst the large bowel felt distended with material of about the consistency of putty, which pitted readily on pressure.

During the next four days the symptoms of pain occurred at short intervals as on the 20th, a little watery fæces being passed on the 22nd and 23rd. The appetite was very poor, the temperature normal, and the pulse averaging about 65. Antispasmodic draughts and a further dose of aloes failed to give any relief.

On the 24th the rectum was quite empty, and an examination revealed a mass of hard material in the large colon; the mass was distinctly felt by all of us, and after a consultation it was decided to operate before the mare got any weaker.

The operating shed consisted of a small greengrocer's stable, one of the stall partitions having been knocked down in order to convert it into a loose-box, and, as it was now about eleven o'clock at night, the light of an ordinary table lamp had to be brought into requisition. Fortunately, plenty of hot water was obtainable, and the instruments, silk, etc., were all boiled in saucepans over the kitchen fire. Several buckets and clean cloths were called into requisition, and cleansed and disinfected with perchloride of mercury.

The animal was cast on the left side, and a large area of the right flank was clipped, shaved, and then covered with wadding soaked in solution of chinosol; this was allowed to remain on the skin for about twenty minutes before an incision was made. Chloroform having been administered, an incision about 6 inches long was made in an almost vertical direction, commencing at a point about 7 inches below the angle of the haunch, and 3 inches away from the costal cartilages of the false ribs. The wound extended to within 2 or 3 inches of the fold of skin which is found underneath the flank. The right hind leg was drawn back, but was not withdrawn from the hobbles. The external oblique muscle was then cut through, the internal oblique being separated with the scalpel handle and fingers as much as possible, with the idea of lessening hæmorrhage; all bleeding vessels were taken up with artery forceps, and the wound from time to time thoroughly cleansed with solution of chinosol (1 in 1200). The peritoneum was incised with a scalpel and director, and after again thoroughly cleansing and disinfecting my arm and hand, an exploration was made for the obstruction. The latter was felt at the termination of the double colon, and appeared to the fingers like a very large mass of hard, dry material; it was clearly what is commonly termed a "dust-ball." Upon attempting to raise it, the mass seemed exceedingly heavy, so much so that I thought I would try the effect of kneading it with the ends of my fingers and knuckles; I did this for about ten minutes, and although hard and unyielding at first, the edges presently appeared to give way, and ultimately the whole mass was broken up into hard, knobby pieces. Upon exploring further along the bowel I did not detect any further lumps of any great size, and therefore decided not to attempt to withdraw or open the gut.

The peritoneum was drawn together with silkworm gut sutures, the muscles and skin being closed with aseptic silk, and the whole covered with a pad of wadding soaked in chinosol solution and held in place by broad calico bandages. The operation was completed about 2.30 A.M.

25th November. The mare got up about an hour later and seemed quite free from pain. She was closely watched and about 8 o'clock drank some water and about a gallon of gruel, and ate a good handful of turnip tops, four carrots, and a quantity of grass. At 10.30 the pulse was 72, temperature 104°, respirations normal. About 11 A.M. she passed about a quart of soft dung containing three or four hard knobs of fæces; this was the first time since the 20th upon which she had passed any solid fæces. At 4.30 P.M. she seemed quite bright and still free from pain; temperature 104·5°, pulse 70. She attempted to urinate, but appeared afraid to stretch herself on account of the pain caused by the wound; the bladder was emptied by the aid of a catheter. At 8 o'clock the temperature was 104·2°, and pulse 68; there was no pain and the animal was quite cheerful.

26th, 27th, 28th, and 29th November. She was quite free from pain and looking bright, the appetite being fairly good and the temperature still remaining at 104° or 104·5°. Internally she received sodæ hyposulph., sod. salicylat. or potass chlorat., in the drinking water, the wound being dressed twice daily with chinosol solution.

On the 27th the external sutures opened somewhat, but the wound was quite sweet and healthy. She passed watery fæces four or five times.

On the 28th the remainder of the skin sutures were taken out and the place treated as an open wound; fæces were passed as on the 26th.

On the 29th the appetite was not quite so good as on the previous days; respirations, too, were somewhat accelerated. Watery fæces were passed twice.

On the 30th the appetite again improved, the wound appearing clean and healthy, and the animal passing fæces four times.

1st December. No fæces had been passed during the night; the animal was not eating and seemed in pain, perspiring freely, and standing with her head fixed in a corner. Temperature was 105° and the pulse too rapid to count. An antispasmodic draught gave no relief, and the owner, without Mr Bell's knowledge, caused the animal to be poleaxed about 10 o'clock.

Post-mortem examination (made by Mr Bell) revealed the presence of fœtid purulent fluid around and in the interstices of the external wound; the internal oblique muscle appeared to have quite united and looked healthy. The parietal peritoneum had healed, and had not the slightest trace of pus on its interior.

The bowel itself, at the termination of the double colon, near its junction with the narrow part of the single colon, showed marked inflammation of the external coat, and upon cutting into it at this part a "dust-ball" was found with a circumference measuring 18½ by 19½ inches, and weighing 6 lbs. It was so situated that it completely blocked up the lumen of the gut. The mass was simply a concretion, and could readily be cut with a knife, the exterior also being easily broken off in small pieces.

Remarks. Laparotomy and laparo-enterotomy are not operations to be undertaken in the horse unless one is absolutely certain of the diagnosis, and takes into account the probable sequel which will ensue if operative treatment be not attempted. It is rather interesting to note that the breaking up of the concretion, or that part of it which was directly obstructing the floating colon, gave the patient freedom from pain; and the question arises as to whether the concretion which was found after death was present in its entirety when I explored the peritoneum, or whether it became partially consolidated, or very much increased, during the week which elapsed after the operation. It may be that the concretion found was the same one which I had broken up, the pieces having again become re-united. I was much struck with the small amount of hæmorrhage which followed the section of the muscles, and the ease with which it was controlled. I see that Messrs Dollar, Rogers, and Rickards also made a special note of the same point when giving the account of a case of laparo-colotomy in the *Journal of Comparative Pathology and Therapeutics* for June 1894.

The cleanliness and healing of the peritoneal wound tends to show that peritonitis is not so much to be feared if the principles of aseptic surgery are observed.

CLINICAL NOTES.

By FRED. HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

RANULA.

DURING the past year two cases of ranula have been observed in the Free Clinique, one occurring in a horse and one in a dog. In each case the animal was brought up with a history of pain in the region of the mouth, accompanied by a certain amount of salivation. In the dog the swelling was about the size of a large hazel nut and situated on the left and under surface of the tongue. In the horse the swelling was quite as large as a goose egg, and also on the left and under surface of the tongue. Treatment in each case consisted in removing as much as possible of the wall of the swelling with scalpel or scissors, and treating the parts with antiseptic lotion. In each case the ranula returned, in the dog after an interval of three months, and in the horse after about two months. Operative treatment was again resorted to, and the wall completely excised; the horse has given no further trouble, and, as it is about four months since, I hope that a cure has been effected; in the dog, however, the last operation was performed on the 4th November, and there seems to be perceptible signs of its re-appearance.

FOREIGN BODIES IN THE MOUTH, THROAT, AND ŒSOPHAGUS; ŒSOPHAGOTOMY.

In all canine practices it is a matter of comparatively common occurrence for cats and dogs to be brought suffering from irritation produced by the presence of some foreign body in the throat or mouth.

The symptoms usually shown are those of pain, particularly demonstrated when the animal is attempting to take food, continual gulping, sometimes great salivation, and occasionally the mouth will be kept partly open as if the patient were gasping for breath. Abscesses may form and the foreign body finds its way to the outside. During the past three years we have had about fifteen or twenty cases of the kind, some of which have had the foreign body removed without difficulty, whilst others have required a certain amount of patience and manipulative care.

By far the most common source of trouble, especially in the cat, has been either a needle alone or a needle and thread; in one case the offending article was situated right at the lower end of the cervical portion of the œsophagus, and was only reached by the aid of a long pair of curved forceps after the cat had been partly anæsthetised with chloroform. The chief caution to be observed is to make sure that the point has been extricated before commencing traction. After needles, fish-bones have been most noticeable, and these, if unable to be removed by forceps, have been either pushed down as far as the stomach or brought up into the mouth by the aid of a probang, similar in pattern to the one used in human practice. Splinters of bird or rabbit bones are treated in the same way.

In one instance, that of a retriever dog, the animal had been noticed by the owner to be uneasy, unable to feed, and salivating a great deal, but it had apparently never occurred to him to examine the mouth. When examined in the Clinique it was found that about 2 inches of the hollow shaft of a sheep's femur had got wedged firmly along the top of the lower molars on the right hand side. After some manipulation this was removed, and the patient gave no further trouble.

Two cases have occurred in which the animals have swallowed large pieces of bone and been unable to pass them through the first two ribs, or the thoracic portion of the œsophagus, into the stomach.

CASE I. was a toy bull terrier pup, brought for treatment, 4th December 1895, because of inability to eat solid food, and general emaciation. The owner stated that the animal had been losing flesh for the past three weeks. No swelling of the œsophagus or foreign body of any kind could be detected, and by the owner's request a toxic dose of hydrocyanic acid was given.

Post-mortem examination revealed a portion of the vertebra of a mutton chop bone (about three-quarters of an inch long and half-an-inch wide) firmly wedged in a dilated sac of the œsophagus about an inch above the stomach; it was really situated in that portion of the œsophagus which lies within the thorax.

CASE II. was a bull dog belonging to Mr Perkins, M.R.C.V.S., of Hastings. The animal was observed to show symptoms of choking on the 14th of October, after picking up something in the garden. Examination revealed a large swelling on the course of the œsophagus just before it enters the thorax. An emetic was administered, lubricants were tried, and instruments were resorted to, but without avail. Œsophagotomy was performed on the 18th, the foreign body proving to be the lower end of a large sheep's femur.

The operation was performed as follows. The dog was placed on his back on the operating table with the throat as much exposed as possible ; after having shaved the parts and disinfected the whole area with a solution of chinosol (1-1000), an incision measuring about 3 inches long was made through the skin directly over the obstruction ; the vessels of the neck were pushed on one side by the aid of the flat part of a scalpel handle and the œsophagus opened. After some manipulation the piece of bone was abstracted ; vomition occurred two or three times during its removal. The pouch formed by the bone contained a quantity of very fœtid material, which was carefully washed out with the chinosol solution. The œsophagus was closed with interrupted sutures of silkworm gut, the muscles and skin being sutured with catgut ; the external wound then received a coating of iodoform and collodion (1-12).

Healing speedily took place without suppuration, and at the end of ten days the dog was sent home with only a slight scar to mark the seat of operation.

No solid food was allowed for two days after suturing the wound, 10 grains of boracic acid being administered twice daily in milk. On the 20th and 21st finely chopped meat was allowed, and after that no further care was taken except to see that no bones or large lumps of meat were given.

Mr Perkins informs me that the animal is now apparently as well as ever, but that if large lumps of food are greedily taken vomition sometimes occurs ; if, however, the food be eaten slowly it is not returned.

FOREIGN BODIES IN THE STOMACH AND INTESTINES—PERFORATION.

CASE I.—8th May 1895. Cat, about three years old, was brought for treatment on account of being an irregular feeder, and suffering from marasmus. Temperature was 103·4°, and, suspecting tuberculosis, the animal was destroyed.

Post-mortem examination revealed the presence of half a wine cork, quite unaltered by the juices of the stomach or intestine, situated in the duodenum, in a dilatation which had become formed to accommodate it.

CASE II.—February 1895. Kitten, about six weeks old, suffering from marasmus. The animal died, *post-mortem* examination revealing several yards of sewing cotton coiled up into a mass in the intestine.

CASE III.—May 1895. Healthy pup, destroyed for dirty habits. *Post-mortem* examination revealed the presence of a glass marble in the stomach.

CASE IV.—August 1896. Fat pug, very fond of crabs. The animal swallowed the greater part of a crab's claw, and suffered severely for three days from gastro-enteritis. The administration of opium and castor oil, together with hot fomentations externally, had the desired effect, and the claw was passed out on the third day.

CASE V.—October 1896. Manchester terrier, brought to the Clinique suffering from gastro-enteritis, suspected to be due to a foreign body. A dose of castor oil caused the expulsion of a plum stone, and the animal made an uneventful recovery.

CASE VI.—Fox terrier poisoned by strychnia. The animal had accidentally obtained access to some poisoned meat. Whilst making the *post-mortem* examination some eight or ten damson stones were observed in the first part of the duodenum.

CASE VII.—1st July 1897. Blenheim spaniel, brought for treatment on account of an abscess in its side. This had burst but the wound refused to heal. Thinking that this was due to a necrosed rib, the latter was being scraped with a Volkmann's spoon, when the point of a needle was discovered. This was taken hold of with forceps and withdrawn together with about 8 inches of thread; it came directly out of the stomach. The animal made a good recovery.

CASE VIII.—16th October 1897. The body of a pug which had died suddenly was brought for examination. Mr Hall, M.R.C.V.S., College Tutor, who made the *post-mortem*, found that the stomach contained a wooden meat skewer, 5½ inches long. Part of it had passed with its blunt end downwards into the intestine, whilst the sharp end had penetrated the wall and become broken off against the diaphragm. The animal must have swallowed the skewer with meat on it at the time of feeding.

A similar case is recorded in the *Journal of Comparative Pathology and Therapeutics* for September 1895, but in that case the point of the skewer came out between the eleventh and twelfth ribs and the animal made a good recovery.

LAPARO-ENTEROTOMY IN A CAT; DEATH FROM CARBOLIC POISONING.

The patient, a female cat in a very emaciated condition, was brought up for treatment on the 12th of August. The animal had been noticed to be unwell for the past three weeks, and had frequently had attacks of diarrhœa and vomiting, but during the past two days had been unable to retain any food even for a few minutes.

Examination through the abdominal walls revealed the presence of a foreign body in the small intestine. Ether was administered and laparo-enterotomy performed. The foreign body proved to be a piece of cork, and unfortunately ulceration and perforation of the intestines at one small place, together with escape of a small quantity of the bowel contents, had already taken place. The edges of the ulcerated and necrosed portion were cut off, the interior and exterior of the bowel together with the peritoneal cavity carefully wiped with carbolic acid solution, and the wound was drawn together with Lembert's sutures. The abdominal wall was sutured in the usual way, and the external wound treated with iodoform and collodion (1-12).

For the instruments and silk I had thoughtlessly been using a 5 per cent. solution of carbolic acid; and, although I had used the same preparation after shaving off the hair to disinfect the skin, and also to wipe the edges of the bowel wound and peritoneum where soiled, I had been very careful to use it as sparingly as possible. However, before the operation was complete symptoms of carbolic acid poisoning had commenced; the voluntary muscular system was twitching violently, and the temperature becoming subnormal. Antidotal measures were adopted, but an hour later the temperature had become reduced to 96°, and within two hours after the completion of the operation the patient was dead. This case is a good illustration of the susceptibility displayed by cats to the effects of carbolic acid.

INJURIES CAUSED BY INDIA-RUBBER RINGS.

On the 10th of November a mongrel terrier was brought by its owner on account of having one hind foot much swollen. This had been observed for about two days. Examination revealed the presence, immediately above the digits, of a small circular wound with clearly defined edges passing in a regular manner around the leg. Upon exploring this with a pair of forceps a small india-rubber ring was detected and readily removed; after-treatment consisted in the application of antiseptics, and the animal made an uneventful recovery.

A similar case occurred in the Clinique during the early part of 1896, the patient being a wire-haired terrier. In this case the animal had a thin india-rubber ring embedded in the skin of its neck, the history being that it was supposed to have been placed there by some children who were in the habit of playing with the animal.

AMPUTATION OF BOTH FORE LEGS OF A DOG.

In July last a valuable whippet dog, eight or nine years old, was brought for treatment on account of having jumped through a window and sustained very severe fractures of both fore legs. Examination revealed compound complicated fractures of the radius and ulna in each case, the ends of the bones protruding through the skin. The injuries had been done several days before, and splints had been applied but with no windows left for the treatment of the wounds, the consequence being that the wounds and tissues below them were becoming gangrenous. Amputation was decided upon as the only means of saving the animal's life, and, at the urgent wish of the owner, performed. The flap method was chosen, the parts being cleansed and rendered aseptic, and chloroform used as the anæsthetic. The wounds healed completely in ten days, and the dog then left the infirmary. Its method of progression was very much like that of a kangaroo, consisting either of a series of hops upon the two hind legs or a kind of slow shuffle. At the present time it balances itself and moves about without difficulty, and has lately satisfactorily performed its duties as a stud animal. False legs have been fitted to the stumps, but the dog seems to prefer to be without them.

A CASE OF CANINE DENTISTRY.

This case was undertaken in conjunction with Mr Edward Moseley, L.D.S., the subject being a Schipperke dog whose teeth had almost all disappeared from old age. The animal possessed four canines and the four carnassial molars, all in good condition and firm; also three loose incisors, which were easily extracted. Chloroform was administered, and rough models of the mouth were taken with modelling composition; from these special trays of pewter were cast and accurate impressions of the jaws made.

The teeth themselves were fitted on platinum plates with a complete palate, the deficiencies in contour of the gum due to absorption being made good with black vulcanite, which gives a very natural appearance (*see* Fig. in Plate III.).

Twenty-four teeth were attached to the plate (six incisors, six molars and premolars in each jaw), it not being thought worth while to restore the small molars at the back of the mouth as they are to a great extent functionless. They were ground down to the proper shape and size from the smallest human incisors made of Ash's porcelain. The cutting edges were protected by thin platinum foil upon which gold solder was fused; this gave an efficient edge and considerably lessened the risk of breakage of the porcelain. The plates were adapted to the mouth exactly as in human dentistry, and they are removed every day to be cleansed.

For the first time or two chloroform had to be used in order that the teeth might be removed or re-inserted, but now the animal submits without any trouble. When inserted for the first time the animal manifested great discomfort for about two hours, but after that he did not offer any objection. Before the teeth were put in the dog weighed 7½ lbs., and looked very thin and poor; all food taken had to be finely minced or in fluid form; now, however, he eats meat and crunches bones without trouble, and although the teeth have only been in for about a month his weight has increased to 9 lbs. 1 ounce.

In general health and appearance, too, the dog is most markedly improved.

AN UNUSUALLY LARGE TÆNIA SERRATA.

A complete specimen of the tænia serrata, measuring 10 feet 8 inches long and containing 465 segments, was obtained on 16th November from a greyhound dog (four years old and weighing 62 lbs.) after the administration of a drachm of tenaline in a drachm of water. The worm was accompanied by two others of the same variety, but the largest of these only measured between 5 and 6 feet.

NOTES ON SOME NEW THERAPEUTIC APPLICATIONS.

By FRED. HOBDAY, F.R.C.V.S., Professor of Therapeutics,
Royal Veterinary College, London.

TENALINE.

TENALINE is a new preparation recently prepared by Messrs Willows, Francis, and Butler from the areca nut, with the object of retaining the tæniafuge alkaloids of Arecaine, Arecaidine, and Guvacine, and getting rid as far as possible of the very toxic principle known as Arecoline.

I have now been able to observe its effects and test its efficacy on rather more than sixty cases, the summary of which is as follows:—

Tenaline is certainly of great value as a vermifuge, especially for tapeworms, in the dog and cat. Its effect is rapid, the average time being about fifteen or twenty minutes; the quickest time in which we observed expulsion of tæniæ after its administration was three minutes, one patient being a spaniel suffering from tænia cucumerina, and another an Airedale terrier suffering from ascarides. It has the advantage over powdered areca nut of being much easier to administer as its bulk is small, and a purgative is not a necessary adjunct. Its effect on ascarides is also well marked, as they may either become vomited up from the stomach or expelled in the fæces.

Its action on the bowels is to increase secretion and stimulate peristalsis, so that even if no worms be present an animal which has received a full dose passes liquid evacuations. Above all, its general effect seems invariably to be to cause expulsion of the head as well as the segments, thus getting rid of the most troublesome part of the parasite.

With reference to dose, that recommended on the bottle is an allowance of 1 minim for each pound of body weight, and it is also advised to be administered pure or with the addition of a little water. The latter we found to give the best results, the addition being made of an equal amount or rather more; that the results are better when water is added, especially where small doses of the medicine are prescribed, may be explained by the fact of it being necessary to give a sufficient quantity of fluid to reach and come in direct contact with the worm which is lying in the intestine.

Although one minim for each pound of body weight is usually sufficient, our observations appear to show that this amount can be doubled with absolute safety; in fact, we have in five or six instances allowed 4, and in one case 8, minims for each pound. The only noticeable effect was vomiting, temporarily increased peristaltic movement of the bowels, accompanied by colic, and the passage of liquid fæces; no after results other than those of dulness and slight diarrhœa.

That tenaline is, however, toxic was illustrated by the case of a Maltese terrier, weighing 4 lbs., and known to be the host of intestinal parasites. One drachm of pure tenaline was given subcutaneously. In ten minutes the animal became unconscious, respirations became very shallow, and five minutes later the dog was dead. *Post-mortem* revealed nothing diagnostic, and the intestines contained living specimens of ascarides and tæniæ.

That it is unsuitable for subcutaneous use was well illustrated by two other instances in which a retriever weighing 35 lbs., and a fox terrier weighing 12 lbs., each received a drachm of pure tenaline subcutaneously. Signs of irritation were marked at the seat of injection for about an hour and a half afterwards. During the following week very tender swellings formed, and on the seventh day that on the fox terrier burst, leaving a jagged ulcerating wound which took some time to heal. The retriever was chloroformed and destroyed; the swelling was found on *post-mortem* to be a large gelatinous mass measuring 5 x 3 inches. In the terrier, for about an hour and a half after the injection there were marked rigors, dulness, and salivation.

With reference to the dose which can be administered with safety to other of our patients, a light van horse received on a fasting stomach 2 ounces of tenaline mixed with 4 ounces of water without any noticeable effect. The animal was killed about four hours later, and *post-mortem* examination revealed a large number of bots in the stomach, also numbers of the strongylus armatus and tænia perfoliata in the cæcum, all being alive and apparently uninjured in any way. A donkey received an ounce mixed with 5 ounces of water, whilst another one received 2 ounces mixed with 4 ounces of water; in the latter case the fæces were passed rather more frequently and of a more laxative character than usual, and the animal was rather dull on the succeeding day.

A heifer received an ounce in 6 ounces of water without any effect.

A full-grown sheep received 2 drachms, and another 4 drachms, each in 4 ounces of water, without effect; whilst on the following day the one which had received 4 drachms received 2 ounces in 6 ounces of water without any noticeable result. A pigeon received 10 minims made into a pill mass with liquorice, and showed no noticeable symptoms of any kind.

THE USE OF BINIODIDE OF MERCURY AS A SUBSTITUTE FOR POTASSIUM IODIDE.

The great objection to the use of potassium iodide in veterinary practice, especially for the larger animals, where full and repeated doses have to be employed, is its expense. About two years ago, in the course of conversation, Professor Edgar of Dartford observed that he had been using the biniodide of mercury as a substitute for potassium iodide in the treatment of actinomycosis, and that he found the results to be attended with an equal amount of success. The recipe used consisted of from 2 to 6 grains of biniodide of mercury dissolved in an ounce of water by the aid of from 5 to 10 or 12 grains of potassium iodide. Since then I have always mentioned the matter to my students when lecturing upon the subject, and I have been able to collect a fair number of instances in which it has been tried successfully on cattle suffering from actinomycosis, especially from Messrs Penhale (of Barnstaple), R. W. Hall (at present one of the College tutors), and Harold Masson (of Flaxton, Yorks.); thus confirming the effects observed by Professor Edgar. The only bad effect that I have had reported was that of a cow which temporarily lost all her hair owing to the medicine being given for an unnecessarily long time, without the necessary occasional visit from the veterinary surgeon in charge of the case, to see how matters were progressing. Knowing the value of potassium iodide in the treatment of scirrhous cord, of elephantiasis of the legs following as a sequel to lymphangitis, and of various tumours in the regions of the shoulder and elbow, I determined to give the biniodide of mercury a trial in these cases in horses.

In four cases scirrhous cords were treated with the object of causing them to become small enough in size for operative purposes, each of them being bilateral and much too diffused to give a fair chance of success until reduced by some means. Two of the owners sold their animals after a fortnight as they would not take the trouble of treatment; the third animal could not trot at all when brought to the Clinique, and only walked with difficulty, but after a month's treatment it was able to go to slow trotting work for three days a week, and was then sold and lost sight of.

The fourth case was a cab horse which had been bought when suffering from a large bilateral scirrhous cord; it was brought to the

Clinique on the 11th of February 1896, and was then absolutely unable to do any work. Two grains of biniodide of mercury, dissolved by the aid of 5 grains of potassium iodide in an ounce of water, were given twice daily in linseed gruel. A week later there was a decided improvement in the way the patient walked, and on 2nd March the horse was sent to slow work.

9th March. The scirrhous cord was perceptibly less, the alterative effect of the medicine was well shown in the animal's improved appearance, and the dose of biniodide was increased to 4 grains twice daily, this being dissolved by 6 grains of potassium iodide.

Treatment was continued until 8th May, the animal now working regularly in a cab, and the cord perceptibly decreasing. On this date the dose of biniodide was increased to 6 grains, 10 grains of potassium iodide being necessary to ensure its solution.

On the 22nd the scirrhous cords appeared small enough to permit of removal by the knife and écraseur, and unfortunately this was attempted. The animal died of shock and collapse two hours later.

In the treatment of chronic elephantiasis and of tumours of the shoulder and elbow we have used the biniodide in the Clinique as a substitute for potassium iodide, and have got an equal amount of success, together with the advantage of obtaining the alterative effect of the mercury. Professor Edgar has recently told me that he has also tried it successfully in the horse for the dispersal of glandular enlargements, for capped elbows, and for scirrhous cord; also that he has found it a valuable agent to use as an astringent to the lacteal secretion in mares and cows, and that on this account it must be used with a certain amount of care in milking animals unless it is desired to arrest the secretion.

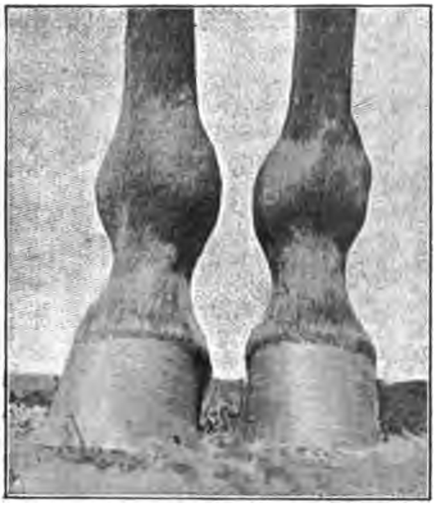
Summing up the results, it must be admitted (1) That the agent is very much cheaper to use than potassium iodide. (2) That the results which I have been able to collect regarding its value in actinomycosis confirm those which Professor Edgar had already obtained. (3) That the observations which Professor Edgar and myself had been independently making at the same time upon its therapeutic effects as a resolvent, specific, and alterative in certain diseases of the horse appear to demonstrate its value, and to agree in almost every detail. (4) We have each found that, as is frequently the case with potassium iodide, failure to continue with the medicine for a sufficiently long time may cause a relapse, the tumours again enlarging.

ANKYLOSIS OF THE FETLOCK JOINT.

By E. E. MARTIN, Vet.-Lieut., A.V.D., Barrackpore, India.

A well-marked case of this affection came under my notice a short time ago, and as I had an opportunity of photographing the legs during life and the bones after death, I thought a short account of it might interest the readers of this Journal.

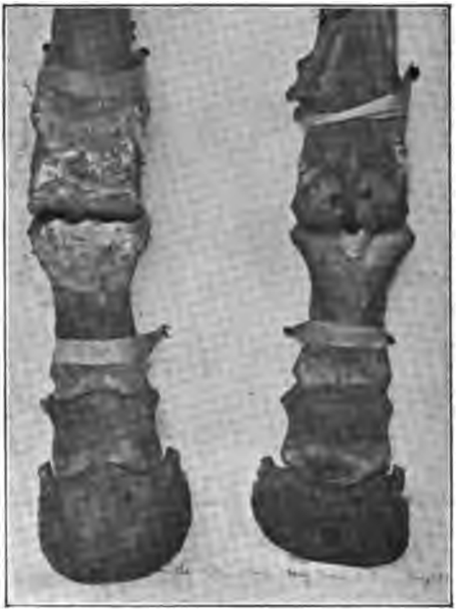
The case was a country-bred chestnut pony mare, aged, height about 13 hands 3 inches. The owner, an artillery officer in this station, had purchased her about a year previous to the time I saw her. At the time of purchase the fetlocks were both rather enlarged and round, but she was going quite sound. He had her examined, and the veterinary surgeon told him that there was a doubt about the fetlocks; she might keep sound, but the condition might get worse. A few months after purchase the enlargement increased, and her gait got stiffer and stiffer. Various applications were tried, and, in addition, she was blistered twice. The condition gradually got worse, and when I saw her first the appearance of the fetlocks was such as is seen in the photo. She was very lame at a trot, slightly worse on the off fore, her action being peculiarly "proppy," just as one might expect from almost rigid fetlocks. She made rather a good canter—in fact at that time the owner was playing polo on her regularly. Examination showed practically entire loss of power of flexion of the fetlock joints, and attempts at flexion were greatly resented as being very painful.



I recommended forcible flexion, and accordingly had her cast and chloroformed, and then flexed the joint until I could get the pastern at right angles to the cannon. The noise made by the snapping of adhesions was very marked. Both fetlocks were flexed in this way. After the operation, when she was permitted to get up, a cotton wool pressure bandage was applied to both joints, in order to prevent any effusion that might take place from the ruptured and strained adhesions. Next day there was considerable improvement in going. Systematic daily flexion was then carried out. A fortnight later she was again cast, chloroformed, and the fetlocks flexed, and this was repeated ten days later. After each operation temporary improvement resulted, but this was not maintained, the joints gradually stiffening again.

After the failure of the flexion treatment, I decided to perform neurotomy. The reason for this decision was that I had seen a case similar to this some time previously, in which both fetlocks were quite rigid, but it was performing its regular work, and beyond a little stiffness in trotting no exception could be taken to its paces. So I came to the conclusion that in these cases lameness is more the result of pain than of mechanical interference. I therefore performed plantar neurotomy on both fore. I regret very much that I did not perform median neurotomy, as I consider it would have been a good case for this operation. However, she derived considerable benefit from this, most of the intense lameness disappearing, and she did her ordinary work for some months. The owner then, having got six months leave, decided to have her destroyed.

The bones were mounted by Farrier-Sergeant T. Gill, and I photographed them. The photo shows the front aspect of the off fore and the back of the near fore leg. It will be seen that the lower portion of the metacarpal bone and the upper portion of the os suffraginis are thickly covered with ossific deposit. This deposit extends upwards as far as the bulbs of the splint bones, which are also involved. The articular faces of the joint were free from the deposit.



Since the above was written *The Veterinary Record* of 2nd October 1897 has come to hand, in which there is a case reported by Vet.-Major Joshua Nunn, entitled "Fibrous Ankylosis of the Fetlock Joint." From what I can gather, his case is similar to the one I have just described, but his was not in nearly so advanced a condition. I think the photograph of the bones may help to throw some light on the pathological conditions of the part, about which he does not venture an opinion. Some details as to the further progress of his case would be interesting, as my experience with the flexion treatment of lameness in the fetlock is that beneficial results are often obtained temporarily, but are very seldom of any permanence.

A CASE OF RUPTURE OF THE BLADDER IN A HORSE.

By W. P. STABLEFORTH, M.R.C.V.S., Assistant to A. JAMES-RIDGWAY, M.R.C.V.S., Honiton.

ON the afternoon of Sunday, the 7th November last, we were requested to go out to a farm in this neighbourhood to see a colt. On arrival I found that the subject was a two-and-a-half years old bay colt which had been taken suddenly ill in a field that morning. The animal had been very restless, making frequent attempts to urinate, and at the time of my arrival the patient was rather dull; the pulse was about 70, full, and fairly strong, and the temperature 101·5°. On examining the bladder per rectum I found that it was greatly distended with urine, and slight pressure on it caused pain to the patient. This suggested an examination of the urinary passage, and on manipulating the prepuce, penis, and perinæum I felt in the urethra, about 8 inches behind the glans penis, a hard swelling about 1½ inch in length by about ¾ inch in breadth; careful manipulation enabled me to detect a small calculus in this swelling, and I at once injected a little linseed oil into the urethra, and with the finger and thumb tried to force the calculus onwards towards the meatus urinarius. These efforts were successful in displacing the calculus in the forward direction for about 3 inches, but beyond that point I could not get it to move; I therefore broke it down with dressing forceps and removed it in pieces. After this I injected into the urethra a five per cent. solution of creolin, and a few minutes afterwards the colt urinated very freely. All the time I had been operating he never moved, although he was not secured in any way.

Before leaving I administered a stimulant, had him made comfortable, and expected to find him greatly improved next morning.

Early on the following morning we had another message saying that the colt was again worse, although he had passed water several times since we left. My preceptor, Mr James-Ridgway, M.R.C.V.S., accompanied me this journey, and we found the colt standing with head depressed in a corner; respirations about 40; pulse very weak, small in volume and about 80 per minute; temperature 103·5°; conjunctiva greatly injected; colt making frequent attempts to urinate, but only a little blood passed. We succeeded without difficulty in passing a catheter, but on withdrawing the stilette got no flow of urine, and on examination per rectum we found that the bladder was completely empty. From these symptoms and the marked depression we suspected uræmic poisoning. We gave a very grave prognosis, and put the patient on milk and linseed gruel; he also had three doses of extract of belladonna during the day, and a lotion was injected three times into the urethra.

The colt died on the morning of the following day, namely, the 9th November, and the *post-mortem* revealed the presence of about two gallons of liquid with a strong urinous odour in the peritoneum. The bladder was ruptured on its lower aspect a little in front of its neck;

it contained a quantity of grit and gravel. The wall of the bladder at the seat of the rupture was very thin. The left kidney was enlarged and somewhat softened; its pelvis contained pus and a number of calculi, the largest about the size of a hazel nut.

SUCCESSFUL SERVICE BY A CASTRATED STALLION.

By T. G. HEATLEY, M.R.C.V.S., Woodbridge, Suffolk.

PROBABLY most people if asked whether a horse that had been deprived of both his testicles could procreate his species would unhesitatingly reply in the negative, and yet the answer ought to be in the affirmative, as the following case will show.

On the 24th of June 1896 I castrated a five-year-old Hackney stallion. Three days afterwards the owner, seeing that the horse was apparently in his accustomed form, presented a mare to him, with the result that she was promptly covered. The mare in question was a Hackney, nine years old, which had had a foal when a three-year-old, but since then had not been covered by any other horse than the above castrated stallion, and by him only on the one occasion, viz., on the 27th of June 1896. Between that date and the 5th of June 1897, when the mare gave birth to a healthy living foal, no horse or colt had access to her. These facts are vouched for by the owner, William Wilson, Esq., Raydon Hall, Orford, Wickham Market.

There is, of course, nothing marvellous in the fact that castration does not instantly deprive an animal of procreative power, for probably in nearly every case living active spermatozoa are left in the vasa differentia and in the vesiculæ seminales. What is really the the most wonderful fact in connection with this case is that the animal still had the sexual instinct as late as the third day after castration, and was so little inconvenienced by the after-effects of the operation that he was not deterred from the act of copulation.

CURIOUS LESIONS OF NAVICULAR BONES.

THE somewhat remarkable navicular bones reproduced in the left-hand bottom figure of Plate III. (facing p. 295) were forwarded by Vet.-Captain Pease, Principal of the Lahore Veterinary College, with the history that they were taken from an old East Indian pony which had suffered from an incurable lameness of both fore feet, simulating navicular disease. In each case the abnormality affects the lower edge of the bone, behind the surface by which it articulates with the os pedis. In the one bone (the uppermost in the figure) this edge shows two rough shallow pits, as if two pieces of bone had been broken off. In an exactly similar position in the other bone this edge carries two little pieces of bone which also look as if they had been broken off: they are now firmly joined to the edge of the navicular by bony union. The appearance of the parts suggests that under strain small pieces of bone giving attachment to the interosseous ligament of the coffin joint had been torn off.

HERMAPHRODITISM IN A PIG.

By JOHN B. YOUNG, M.R.C.V.S., Braintree, Essex.

A SHORT time ago I called at a farm to castrate and spay a number of pigs about eight weeks old. Before I commenced to operate the owner informed me that there was something wrong with two of them belonging to the same litter, both urinating like a female yet having the sheath and one of them the testicles of the male. Upon making a closer examination I found that one had the external organs of both sexes, viz., vulva, testicles, and sheath, whilst the other showed no testicles externally. The vulva in both was about half an inch long, and hung down like a small penis ; the urinary opening was on its upper surface, and closely resembled the meatus urinarius of the mare. The sheath was of normal size for male pigs of that age, excepting that there was no urinary passage.

I first operated upon the one not having testicles visible, and after removing the uterus found a perfect testicle of the usual size, attached at each extremity in place of ovaries. The testicles in the other were removed, but no uterus could be felt when I operated on it for the female operation.

I consider a pig with testicles attached to the extremities of the uterus, and externally having the sheath of the male and vulva of the female, about as perfect a hermaphrodite as one often meets, although, whilst at College I was told a perfect one should be able to beget, as well as bear its kind.

SALIVARY CALCULUS.

By E. LIONEL STROUD, M.R.C.V.S., Colchester.

As cases of salivary calculus are somewhat uncommon, the following one may be of interest.

Whilst attending a case of poll-evil, with my partner, Mr Prime, our attention was directed to an aged bay cart mare, which had a swelling on the side of her face.

History. The owner had bought the mare three years previously ; she had then a swelling about the size of a walnut, which would break out and discharge for a time, and then heal up only to break out again. This had been going on up till the day we saw her, the swelling at the same time gradually getting larger.

Symptoms. The swelling was hard and slightly movable, little or no pain being caused by manipulation. Its position was on the outer surface of the under jaw, at about the level with the third molar tooth. There was slight discharge, which was decreasing daily.

Treatment. After washing the part, a longitudinal incision about 2 inches long was made from the centre to one end of the swelling, when the calculus was easily removed. The wound was treated by plugging with digestive ointment on tow. This was continued until the hole became too small to introduce a plug, after which astringent lotion was applied and the granulations cauterised. There was a flow of saliva at first, but the wound gradually healed, and was perfectly closed in twenty-three days.

The calculus when removed was found to weigh but a grain or two under 7 ounces; it is greyish in colour, oval in shape, and about the size of a full-sized turkey's egg, measuring in circumference longitudinally $8\frac{1}{16}$ inches and transversely $5\frac{5}{8}$ inches. The side in apposition with the jaw is flattened and comparatively smooth; the rest of the calculus is roughened, and at one end there is a slight depression.

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